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REVISION SERIES

XII. COMMON DISABILITIES OF THE FEET

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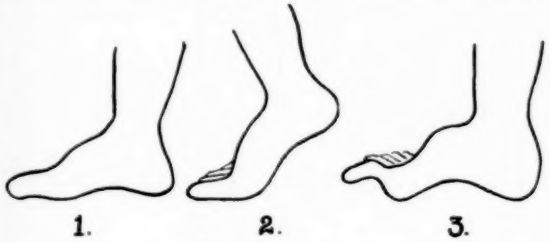
Napoleon may have declared that an army marches on its stomach, but in more recent wars we have realized that a soldier marches with more agility on good feet. Moreover—in time of peace—vigour, good nature and good temper depend on pedal comfort. Osler, with due authority, wrote, 'A man is as old as his arteries', but we may endorse Lewin's addendum, 'but he acts as old as his feet and legs'. It is also permissible to generalize that either the shoe must be made to fit the foot, or the foot must be made to fit the shoe. With that proviso a strong and supple foot is a good foot even if it is flat or unshapely. In other words, if the foot has an abnormality of shape we must either make a shoe that accommodates the abnormality or, by surgery, we must trim the shape to fit an ordinary shoe. If the patient has a shoe which fits the foot or if he walks barefoot, he will be comfortable as long as the foot is strong and supple, whatever its shape.

The Action of the Foot in Standing and Walking

When *standing* on the normal foot, weight is borne on the heel and on the heads of the 1st and 5th metatarsals. This 3-point weight-bearing is converted into a tripod by longitudinal and transverse arches (Fig. 1).

When *walking*, weight is borne alternately on this tripod and on a platform formed by the heads of the metatarsals and the 5 toes in front of them (Fig. 2).



Figs. 1, 2 and 3

In order to stand and walk comfortably, it is essential that the toes should be held straight so that weight is distributed evenly on a broad surface. The toes are straightened by the short muscles of the feet, chiefly the lumbricals and interossei. The arches are supported by the short muscles and also by the long calf muscles.

FOOT STRAIN

In our well-shod and motor-car age the short muscles of the feet are not always sufficiently exercised and tend to weaken. They then fatigue on standing or walking and the toes, instead of remaining a straight platform, claw in the shoes. Weight is borne unevenly on the heads of the metatarsals and the tips of the toes, and the *clawed toes* crowd the shoes. The consequence is metatarsal callosities, corns on the toes and metatarsalgia (Fig. 3). This process is accelerated and exaggerated if the short muscles are injured or are paralysed by poliomyelitis, spina bifida or other nerve lesion.

When fatigue is excessive or repeated, the feet swell towards the evening. After a night's rest the swelling subsides, but if this process is not checked, in due course oedema persists overnight and later chronic oedema organizes. Fibrin is formed, which in turn is converted into fibroblasts and fibrous tissue, and so gradually chronic strain produces increasing stiffness of the joints of the foot and the patient feels discomfort on forced passive movements.

Sometimes, though rarely, the arches of the foot collapse acutely and dramatically. More usually, the arch drops slowly and the fall is accompanied by the formation of osteophytes on the dorsum of the tarsal joints. The most common site is in the neighbourhood of the long extensor tendon to the big toe. When the arch flattens, these osteophytes are squeezed out on the dorsal edges of the mid-tarsal joints. This phase is denoted by tenderness of the spring ligament and by pain under the longitudinal arch. As the short muscles fatigue, the long muscles of the calf try to compensate.

When they tire, aching in the calf becomes the predominant symptom.

Treatment

The treatment of foot strain includes the treatment of symptoms which frequently follow in sequence, viz.: aching on fatigue, metatarsalgia, corns, aching under the longitudinal arches of the feet, and aching in the calves on walking. All are finally aggravated when the feet swell and become stiff. The approach to treatment depends on the stage of strain which has been reached and particularly on whether the feet are supple, are mildly stiff with pain on passive movements, or are very rigid.

(*a*) *Supple feet* can be restored to comfort and vigour by correct proportions of rest and exercise. In severe foot-strain rest should consist of a few days in bed. In less severe instances it may be achieved by limiting activity with or without foot supports. In the early stages the usual longitudinal arch-supports are rarely required. All that the patient needs is a metatarsal pad or platform. This relieves the heads of the metatarsals from full weight-bearing and helps to straighten the toes. When unnecessary, longitudinal arch-supports would weaken the foot and in many instances the patient cannot again do without them.

Exercise should initially be non-weight-bearing and directed to strengthening the short muscles of the feet, the muscles that straighten and separate the toes. The patient is instructed to do the exercises for short periods, 2 or 3 times a day when seated. The traditional direction to walk on the outer borders of the feet serves no purpose, as only the muscles of the calf are so exercised. When the patient can actively straighten and separate the toes good progress has been made. Longitudinal arch-supports are prescribed for cases of long standing and of marked weakness or wasting only. They should be made to measure of resilient, not rigid, material.

(*b*) *Rigid Feet.* In comfortable shoes, rigid feet, though weak, are often painless. If not, the prime requisite is a pair of firm and custom built supports. The supports must be made from a plaster-of-paris cast, for it is seldom that stock sizes are adequate. If standard shoes do not fit, special shoes must be made or surgery will be necessary to reshape the foot. Physiotherapy should aim only at reducing swelling and maintaining the circulation.

(*c*) *The Semi-rigid Foot.* When the patient complains of pain on forced passive movements, a clinical decision must be made. Either the feet must be mobilized by physiotherapy and manipulation and then treated as supple strained feet as in (*a*), or we must accept the disability and treat as a rigid foot as in (*b*). The largest group of 'foot sufferers' fall into this category, but most of them can be completely relieved by the judicious use of manipulation under general anaesthesia, adequate rehabilitation and appropriate supports.

CORNS AND CALLOSITIES

Corns are caused by intermittent pressure over a limited area of skin where there is firm resistance underneath. They occur chiefly on the toes, and under the heads of the metatarsals. It is said that no other minor condition causes as much discomfort, pain and disability as a corn. The frequency with which great writers comment on them is evidence of their prevalence, and Jonathan Swift's line, 'A coming shower your shooting corns presage', describe their association with weather prophecy.

Callosities follow intermittent pressure over a wider area where the underlying resistance is not as firm. They are found chiefly on the soles of the feet, the palms of the hands and the backs of the heels. Callosities may or may not be painful and are often protective.

The conditions which provide the medium conducive to the formation of corns and callosities are: (1) badly fitting shoes, (2) foot strain with its incident wasting of muscle and clawing of toes and (3) deformities of the feet and toes.

Treatment

Prevention of these 3 conditions is the best treatment. Today shoes are manufactured in many sizes with a wide range of fittings and it is easier to find well-fitting shoes. Symptoms of foot strain need early attention and occasionally bony deformities such as clawed toes, hammer-toes, overlapping 5th toes, exostoses and prominent heels, must be corrected surgically.

Once the corn is established, treatment is virtually in the province of the chiropodist, who treats them by pressure-relieving pads, by paring or by removal. Various applications help to soften corns so that they shed themselves. The best of these is a paint of salicylic acid in collodian. It is applied daily until the corn peels off with the 'sticky skin' formed by the repeated applications.

Allied to chiropody we must see that the feet are strengthened, that friction points are eliminated, and that the toes are kept straight, even if to do so manipulation, tenotomies, or surgery are required.

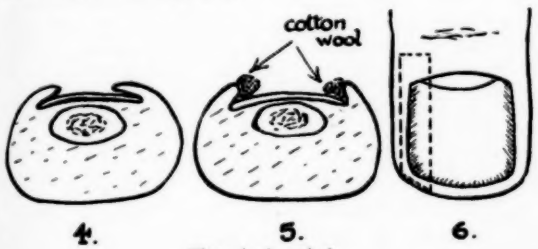
PLANTAR WARTS

A plantar wart is an infective papilloma on the sole of the foot. It does not usually protrude beyond the surface and tends instead to burrow into the soft tissues of the sole because it is covered by a thick layer of cornified skin. It is generally thought to be due to infection by a virus and to be contagious, spreading to the feet of others. It causes disability, not only by the pain it produces but because it is apt to multiply on the foot. It may be difficult to distinguish from a corn but, whereas a corn is tender on direct pressure, a wart is painful when it is squeezed and when pressure is released. Often little black, blue or red spots can be seen under the cornified layer of skin, and a wart should be suspected if it is in an unusual place, where pressure does not normally occur, e.g. under the arch of the foot. As is evidenced by the many household, patent and medical remedies, plantar warts are often difficult to treat. Moreover, results of treatment are difficult to assess, for warts sometimes disappear spontaneously. One can achieve success in many cases by applications of formalin or acetic acid. Surgical methods include coagulation with cutting diathermy. This is relatively

painless afterwards, although the craters heal slowly. Adequate curettage under local anaesthesia followed by packing with permanganate crystals or Friar's balsam is another measure which may be recommended. Deep X-ray therapy should be prescribed only rarely and very cautiously. It does harvest its percentage of cures but, when deep X-ray fails, treatment becomes so much more difficult because of the avascular scar with which it surrounds the wart. Surgery then becomes protracted because the resultant wound is indolent in healing.

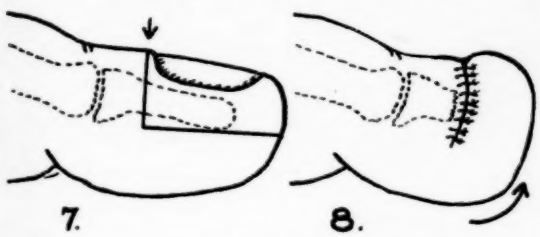
INGROWN TOENAILS

Lewin defines an ingrown toenail as 'one in which one or both lateral margins are embedded in the soft tissues of the nail-groove, producing inflammation'. Usually the big toe is involved and most frequently the cause is cutting the nail too short. Sometimes the shape of the nail is responsible (Fig. 4).



Figs. 4, 5 and 6

In the early stages wisps of cotton wool dipped in Friar's balsam packed between the nail and the nail-fold are all that is necessary (Fig. 5). They open up the groove and relieve the inflammation and pain. Granulating areas should be cauterized with sticks of silver nitrate or copper sulphate. If the condition is more established the affected quarter of the nail with its nail-bed and overlapping flap of skin should be excised



Figs. 7 and 8

(Fig. 6). If the big toe is longer than the 2nd, amputation of half the terminal phalanx and the whole nail-bed, using a plantar flap to cover the stump, may be confidently recommended. The only disadvantage is the cosmetic effect of the loss of the nail (Figs. 7 and 8).

METATARSALGIA

This term is used to describe all aches and pains associated with the ball of the foot. The common forms are due to foot strain, clawing of the toes, and pes cavus. Treatment of the causal factor plus metatarsal platforms or metatarsal bars is often successful in brir ging relief.

A severe type, also known as Morton's metatarsalgia, is due to a neuritis of the plantar interdigital plexus of nerves. The plexus is formed at the junction of the medial and lateral plantar nerves and is usually situated at the distal end of the third inter-metatarsal space. Occuring most often in women, the classical description is of acute shooting pains or cramps extending into the 3rd and 4th toes, which make her stop walking, remove her shoe and squeeze her foot. After a rest she can continue walking. On examination the plexus is tender, and clicks on squeezing the metatarsals. After a time sensory changes can be detected in the affected toes.

In the early stages the attacks may be relieved by wider shoes, metatarsal platforms and physiotherapy, but excision of the nerve plexus brings a dramatic cure.

HALLUX VALGUS

This is the commonest deformity of the feet to cause disability (Fig. 9). In children it is associated with metatarsus primus varus or adduction of the 1st metatarsal. Osteotomies and muscle transfers are necessary to correct and maintain correction of the deformity, and should be undertaken to prevent increasing discomfort and unsightliness.

In adult life the deformity develops mainly in women

Fig. 9

and is associated with splaying of the foot and flat-foot. The condition may also be complicated by foot strain, by metatarsalgia, by clawing of the toes and corns, by overlapping 2nd toe and, most commonly and almost universally, by an exostosis with bunions on the head of the metatarsal. The bunion becomes inflamed and even infected. Later osteo-arthritis develops in the metatarso-phalangeal joints. Pain which is felt later is due mainly to the bunion or to the osteo-arthritic joint.

Treatment

Some patients make walking tolerable by wearing open sandals or cutting a hole in the shoe over the bunion. In early cases, and especially in young people, corrective appliances and exercises to strengthen and tighten up the feet are helpful. When deformity is established these measures are useless, but surgical correction may be recommended confidently. In my experience a modification of the Kellars operation in which the base of the proximal phalanx and the exostosis on the head of the metatarsal are excised gives very satisfactory results. Thirty degrees of movement with active control should be achieved. This result depends on careful and systematic post-operative care.

The Mayo operation, in which the head of the metatarsal is excised, tends to weaken the tread, and simple excision of the exostosis and bursa, though temporarily comfortable, usually leads to a recurrence of pain and the need for the more radical operation a few years later. When present, complications such as foot strain, clawed toes, hammer-toes, etc., must be dealt with at the same time.

HALLUX RIGIDUS

Hallux rigidus is due to arthritis of the metatarso-phalangeal joint of the big toe, which in these cases is often abnormally long. The frequency with which arthritis occurs in this joint is associated with the fact that it takes more strain than any other in the body—with every step. The patient suffers increasing pain and stiffness and tries to reduce discomfort by walking on the outer border of the foot, which may in turn become painful. And often the presenting symptom when the patient consults his doctor is pain on the outer side of the foot.

The conservative approach to treatment is to protect the joint from movement. This is achieved by wearing shoes with a thick sole and inserting a 'roller bar' between the layers of the sole at the level of the metatarso-phalangeal joints, thereby trying to imitate the effect of wearing a wooden sabot. Where there is much pain the sole is made more rigid by a thin strip of light metal.

Treatment by operation is simpler and should be advised for all fit patients. Again the modified Kellars seems to give the most consistent results.

Most of these disabilities of the feet are preventable if proper attention is given to shoe fitting and to Lewin's 'pedigram', 'Fatigue is a warning; pain is a danger signal; swelling may be serious'.

REFERENCES

Lewin, P. (1947): *The Foot and the Ankle.* Philadelphia: Lea and Febiger.
Lake, N. C. (1945): *The Foot.* London: Baillière, Tindall and Cox.
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NATIONAL GENERAL PRACTITIONERS' GROUP

The following resolutions were taken at a meeting of the Steering Committee of the National General Practitioners' Group, held on 13 April 1955:

'The Steering Committee wholeheartedly agrees with the interpretation of the referendum by Federal Council as follows:

"(*a*) A majority of voters favoured the retention of some sort of registration.

"(*b*) A majority favoured a register of consultants.

"(*c*) A majority favoured statutory registration as opposed to voluntary.

"(*d*) An overwhelming majority held that domiciliary visits should not be made by specialists or consultants except in consultation or in the case of emergency."

'The Steering Committee is most disturbed that Federal Council did not recommend to Medical Council or the Minister that action should be taken to amend the Act in accordance with the wishes of the profession as expressed in the referendum.

'The Steering Committee furthermore calls upon its Branches and Sections to hold meetings of general practitioners throughout the country to pass resolutions calling upon Federal Council to carry out the above resolutions.'

INTRACARDIAC SURGERY UNDER DIRECT VISION

The American College of Chest Physicians, Northern Chapter, South Africa, held their Annual Dinner and 24th Meeting at the Wanderers Club on 24 May 1955. The following new office-bearers were elected for the coming year: President, Dr. A. Rabinowitz; Vice-President, Mr. D. I. Adler; Secretary and Treasurer, Mr. D. N. Fuller; Additional Member, Dr. L. D. Erasmus.

The newly elected President, Dr. Rabinowitz, took the chair and after a few words of appreciation called on Prof. J. K. Bremer who delivered an address on *Intracardiac Surgery under Direct Vision.* Prof. Bremer emphasized the desirability of operating on the open heart under direct vision rather than by blind and indirect procedures. The attempts to make open operation possible revolved around (1) pump oxygenators and (2) the lowering of the metabolism of the body. The advantages and disadvantages of various pump oxygenators to replace the cardio-pulmonary apparatus in open operation were discussed.

Prof. Bremer then discussed in some detail the 'cooling method', by which the reduction of metabolic requirements can be effected to such an extent that cessation of the circulation can take place without danger to the vital organs. Various means of increasing the time available for the operation and of eliminating practical difficulties were outlined. Future developments for perfecting the technique of open operation would probably be along the lines of controlling ventricular fibrillation and perfusion of brain and heart.

South African Medical Journal

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VAN DIE REDAKSIE

ENDEMIESE SKILDKLIERGESWEL IN SUID-AFRIKA

Op bladsy 617 van hierdie uitgawe verskyn 'n uittreksel van 'n verslag oor *Endemic Goitre in the Union of South Africa and some Neighbouring Territories*. Die verslag is opgestel deur 'n Komitee wie se aanstelling deur die Unie Departemente van Gesondheid en Voeding, die Universiteit Pretoria en die Raad vir Wetenskaplike en Industriële Navorsing goedgekeur is. Die verslag bevat waardevolle inligting oor die geografiese voorval en die oorsaak van kropgeswel in Suid-Afrika. 'n Kaart (wat in ons uittreksel verskyn) toon die streke aan waar endemiese kropgeswel voorkom wat volgens die Komitee se mening hoofsaaklik aan 'n jodiumtekort te wyte is en ander streke veral in Noordwes-Kaapland in die omgewing van Kenhardt, Gordonia en Springbok waar dit volgens hul mening veroorsaak word deur te veel fluor in onderaardse waters. Die rol wat fluor speel in die ontstaan van kropgeswel is nog nie beslis nie.

Oor die algemeen kom die jodiumtekort in die bergstreke van die ooste en die suide voor en dit word aanvaar dat die reent die jodium uit die grond geloog het soos dit die geval is in baie ander bergagtige dele van die wêreld. Dit is egter opmerkenswaardig dat kropgeswel in sommige dele voorkom wat nie bergagtig is nie soos in die Wes-Transvaal en veral die Kaprivistrook. In hierdie streke het die Komitee tekens van 'n primêre jodiumtekort gevind maar hul maak melding van die moontlikheid dat antiskildklierstowwe of faktore (insluitende 'n gebrek aan higiëne) miskien 'n jodiumtekort in die hand kan werk as die opname van jodium alreeds uiters laag is. Die oorsaak van die endemiese kropgeswel in die Kaprivi-strook is 'n vraagstuk wat vir 'n lang tyd al aandag geniet. Die mening is al uitgespreek dat sekere neute wat op groot skaal in daardie gebied genuttig word 'n antiskildklierstof mag bevat, maar hierdie teorie word deur die Komitee verwerp.

Die Komitee het gevind dat die jodium-inhoud van die water in die aarde in baie van die Weskaaplandstreke, waar endemiese kropgeswel voorkom, meer as hoog genoeg is en hul postuleer dat fluor as 'n teenskildklierstof ageer. Teoreties is dit moontlik aangesien fluor 'n groter affiniteit vir die tiroksienmolekuul besit as jodium en derhalwe die neiging besit om die jodium te verplaas. Hierdie probleem is egter nog nie opgelos nie aangesien daar streke in die wêreld is waar die

EDITORIAL

ENDEMIC GOITRE IN SOUTH AFRICA

On page 617 of this issue an abstract is published of the report on *Endemic Goitre in the Union of South Africa and some Neighbouring Territories* by a Committee in the appointment of which the Union Departments of Health and Nutrition, the University of Pretoria and the Council for Scientific and Industrial Research, were concerned. This report gives valuable information, which had not been readily obtainable previously, on the geographical incidence and causation of goitre in South Africa. A map (reproduced in our abstract) shows those areas in which endemic goitre, in the opinion of the Committee, is caused by primary iodine deficiency, and other areas, notably the North West Cape around Kenhardt, Gordonia and Springbok, where in the opinion of the Committee it 'is caused by excessive quantities of fluorine in subterranean waters'. It is, however, doubtful whether the causative role of fluorine has yet been finally established.

The areas of iodine-deficiency goitre in general correspond with the mountain belt of the east and south in which, in common with many other mountainous parts of the world, it is assumed that iodine is leached out of the soil by rain. It is noteworthy, however, that goitre occurs also in some areas which are not mountainous, such as the Western Transvaal and particularly the Caprivi Strip. The Committee finds evidence of primary iodine deficiency in these latter areas but suggests the possibility that antithyroid substances or factors (including lack of hygiene) may precipitate iodine deficiency on marginal intakes. The reason for endemic goitre in the Caprivi Strip has long been a subject of interested speculation and suggestions have been made that certain nuts which are extensively consumed in that area might contain an antithyroid substance. The Committee rejects this theory.

With regard to the incidence of endemic goitre in the Western Cape the Committee finds that the iodine content of the water and soil in many of the areas affected is more than adequate and postulates that fluorine acts as an antithyroid substance. This is indeed theoretically possible in that it has been shown that

fluor-inhoud van die watervoorrade te hoog is maar die toestand nie met kropgeswel gepaard gaan nie. Leone *et al.*[1] beskryf so 'n streek en die Komitee maak melding van 'n soortgelyke streek in Maldon, Essex en hul meen dat die afwesigheid van kropgeswel in hierdie streek te danke *mag* wees aan die hoë jodiuminhoud van die drinkwater en aan die vis wat geëet word. Dieselfde verklaring geld moontlik vir Ysland waar fluorose voorkom maar nie endemiese skildkliergeswel nie.

Wat voorbehoeding en behandeling betref, beveel die Komitee die jodisasie van sout aan vir die kropgeswel, insluitende dié in die Kaprivi-strook, wat aan tekorte te wyte is. Ons lesers is miskien nie almal bewus van die feit nie dat jodisasie van sout die praktiese moeilikheid oplewer dat jodiede slegs by droë of geraffineerde sout gevoeg kan word. Die growwe kombuissout trek water en kan nie met welslae met jodiede behandel word nie. Dit is egter onlangs bewys dat jodate met veiligheid by sulke higroskopiese soute gevoeg kan word en dat dit net so doeltreffend is. Deeglike proewe wat onlangs in Guatemala[2] uitgevoer is het deurgaans gunstige resultate getoon.

In die streke waar die fluor-inhoud van die water hoog is, is die Komitee van mening dat die byvoeging van jodium nie die kropgeswel sal uitskakel nie en dat dit nodig sal wees om die fluor uit die water te verwyder. Dit sal vanselfsprekend van die grootste waarde wees as hierdie metode uitgetoets kan word en die resultate oor 'n lang tydperk bestudeer word.

Die verslag bevat waardevolle inligting met inbegrip van die omvattende besprekings oor die verband tussen jodiumtekort en vitamientekorte, en die verskeie tipes stowwe wat kropgeswel mag veroorsaak. Dit is 'n groot bydrae tot die bevordering van gesondheid in ons land.

1. Leone, F. L. *et al.* (1954): *Medical Aspects of Excessive Fluorine in Water Supply*. U.S. Publ. Hlth. Rep. No. 69, p. 925.
2. Ann. Rep. (1953-54) Inst. Nutr. Centr. Amer. Panama, Guatemala.

fluorine has greater affinity for the thyroxine molecule than iodine has and might therefore tend to displace iodine. The last word on this subject has, however, not been said, since there are areas in the world where excessive fluorine in the water supply is not associated with goitre. One such area is described by Leone *et al.*[1] The Committee itself quotes a similar area in Maldon, Essex, and suggests that the absence of goitre there *may* be due to a large amount of iodine in the drinking water and the consumption of fish, which has also been suggested as the explanation of the presence of fluorosis and the absence of endemic goitre in Iceland.

With regard to treatment and prevention the Committee recommends the almost universally supported iodization of salt for the deficiency group of goitres, including those of the Caprivi Strip. Our readers may not all be aware that there is a practical difficulty in the iodization of salt, namely that iodides can only be added to dry or refined salt. The coarser cooking salts which are hygroscopic cannot successfully be treated with iodides. It has recently been shown, however, that iodates may be added safely to such hygroscopic salts and that they are equally effective. A recent considerable trial in Guatemala[2] reports uniformly favourable results.

In areas where the fluoride content of the water is high the Committee feels that the addition of iodine will not abolish goitre and that it will be necessary to remove fluorine from the water. A trial of the latter method with a long-term follow-up would obviously be of great value.

The report itself is full of valuable information, including a comprehensive discussion of the relationship of iodine deficiency to vitamin deficiencies and of the different types of goitrogenic agent, and constitutes a valuable service to the cause of health in South Africa.

1. Leone, F. L. *et al.* (1954): *Medical Aspects of Excessive Fluorine in Water Supply*. U.S. Publ. Hlth. Rep. No. 69, p. 925.
2. Ann. Rep. (1953-54) Inst. Nutr. Centr. Amer. Panama, Guatemala.

CORTISONE AND TENDON HEALING

'In traumatic and reconstructive surgery one of the main problems is the restoration of the gliding action of a tendon after injury'.[1] This is especially important in the upper limbs, where the 'maintenance of mobility in the complicated and vulnerable system of the tendons of the hand'[1] is essential. Connective-tissue adhesions are prone to develop, partly as a normal healing process and partly as an unduly pronounced reaction to trauma. In an important experimental investigation, the results of which were recently published, Nils Carstam[1] has studied the effect of cortisone on the formation of tendon adhesions and on tendon healing. In tendon injuries, whether there is loss of continuity or not, the process of inflammation-repair is chiefly seen round the tendon—in the epitenon and paratenon—with the formation of adhesions.

In previous attempts to prevent adhesion formation the greatest advance was the work of Sterling Bunnell with his so-called 'atraumatic technic'.[2] Bunnell stressed the importance of delicate handling of tissues and the prevention of dehydration, and he introduced new suturing methods. Amongst the other surgeons in this field are Pulvertaft, Koch, Mason and Allen, whose work has also improved tendon transfers and free tendon grafts. The antibiotics have naturally also brought about considerable improvement in the results. Despite these advances, however, the reaction of the tissue to injury is often sufficient in itself to lead to adhesion formation with impaired function of the tendon. Other workers have tried the interposition of organic and inorganic materials to prevent adhesion formation: autografts of fat and fascia have been used, while inorganic substances like cellophane and tantalum foil have also been tried. Polythene was found to decrease the formation of adhesions round sutured tendons, and healing did occur, provided the limb was immobilized for a sufficient length of time. This immobilization is necessary because the polythene

tube blocks the ingrowth of connective tissue and can actually prevent healing if blockage is complete.

Treatment with cortisone causes delay in the healing of primary and secondary wounds in the skin, and topical application of adrenocortical extract also seems to impair wound healing. Moreover, in animals the formation of peritoneal adhesions produced by different means can be suppressed by the administration of cortisone. The healing of fractures, experimental arthritic changes, the formation of granulation and fibrosis round foreign bodies, can be inhibited by cortisone.

Carstam set out to investigate in the rabbit the influence of cortisone upon the healing of tendons and upon the development of adhesions after injury. As a result of his experiments he concluded that (*a*) the parenteral administration of cortisone to rabbits suppressed the formation of adhesions round a traumatized extensor hallucis longus tendon. The dosage of cortisone was important and also the stage at which therapy was instituted. The best results were obtained when cortisone therapy was begun 3 days before operation. (*b*) Cortisone in a dosage large enough to suppress adhesion formation round a traumatized extensor hallucis longus tendon of one leg did not appreciably influence healing of a contralateral sutured Achilles tendon, provided the tendon could be properly immobilized in a position of relaxation.

It has been shown experimentally that the topical application of cortisone adversely influences the proliferation of connective tissue. Clinically the local effect of cortisone has been utilized successfully, particularly in the treatment of eye diseases. Hollander, Brown and Jessar[3] injected both cortisone and hydrocortisone intra-articularly into rheumatoid and osteoarthritic knee joints, and found hydrocortisone far superior to cortisone. The cortisone often failed to produce a response and on occasions proved to be irritating. In Carstam's experimental series he found that the topical commercial cortisone acetate did not diminish adhesion formation round a tendon and caused persistent inflammatory irritation. This was also present when the vehicle alone was used. He did not use hydrocortisone topically, and he is at present engaged on an investigation using hydrocortisone.

Carstam concluded that where tendon suture is required cortisone should be used with caution and only in cases where the limb can be immobilized with the tendon relaxed. This is in order to avoid the risk of impaired healing and the early separation of the tendon ends. However, in reconstructive surgery not requiring tendon suture, e.g. tendolysis, the depressive effect of cortisone on tissue reaction might be utilized with advantage in inhibiting the re-formation of tendon adhesions.

1. Carstam, N. (1953): Acta chirurg. Scand., *Suppl.*, p. 5.
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MEDICAL FEES UNDER THE WORKMEN'S COMPENSATION ACT

The tariff of fees was drawn up soon after the Workmen's Compensation Act was passed in 1941. Since that time great economic changes have taken place, in consequence of which medical fees generally have increased in accordance with the rise in wages and salaries and in the cost of living. The fees payable under the Act have lagged far behind this general increase, and for a considerable time the Association's Workmen's Compensation Act Subcommittee has been negotiating with the Workmen's Compensation Commissioner in order to secure a proportionate increase in the tariff.

The Commissioner attended the meeting of the Federal Council which was held in October 1954, and discussed an offer he had made to institute a 25% increase in the schedule. This offer was accompanied by a proviso that no further changes should be made in the tariff for 3 years, which would give the Commissioner the opportunity of seeing how the changes would affect the Fund. Federal Council accepted the increase on behalf of the Association, on condition that an attempt be made to have the schedule completely revised within the 3 years. The 25% increase came into operation at the beginning of 1955.

The Government of Southern Rhodesia has now published a tariff of medical fees under their Workmen's Compensation Act, and an abstract of the tariff is published in this issue (page 620). The general level of the fees is substantially higher than in the schedule under which doctors are working in the Union. For instance, the Southern Rhodesian fee for a visit (non-Native) is 15s., as compared with 9s. 4½d. (7s. 6d. increased by 25%) in the Union; the difference is less if the patient is a Native. Our neighbours' tariff will be scrutinized with great interest in the Union, and will doubtless be taken into consideration both by the Workmen's Compensation Commissioner and the Association in framing the new tariff for the Union.

WINTER SEMINARS AT GROOTE SCHUUR HOSPITAL, CAPE TOWN

The Department of Anaesthesia, University of Cape Town, announce that seminars, open to all members of the medical profession and all medical students, will be held as follows throughout the winter in the Small A-Floor Lecture Theatre at Groote Schuur Hospital, at 8.15 p.m.:

6 June. Professor M. van den Ende: *The Postgraduate Student.*

20 June. Professor A. W. Sloan: *Modern Methods of Cardiac Investigation.*

4 July. Professor J. H. Louw: *Current Concepts in the Surgical Treatment of Gastric and Duodenal Lesions.*

18 July. Professor R. Turner: *Information required of the Anaesthetist in relation to Deaths occurring under the Influence of an Anaesthetic.*

1 August. Professor J. T. Louw: *Indications for the Termination of Pregnancy.*

15 August. Dr. F. Ames: *Clinical Aspects of Cerebral Anoxia,* with two films.

29 August. Professor T. W. Price: *South African Law relating to the Practice of Anaesthesia.*

MYOCARDITIS OF THE NEWBORN

AN OUTBREAK IN A MATERNITY HOME IN SOUTHERN RHODESIA ASSOCIATED WITH COXSACKIE GROUP-B VIRUS INFECTION

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In late February and early March 1954, 3 babies developed an acute febrile illness soon after birth in a maternity home in Southern Rhodesia. One of them died on the 12th day after birth, and the other 2 recovered after being ill for about 1 week. As this outbreak was found to be associated with an infection with a Coxsackie group-B virus, the details of the clinical findings and the laboratory studies are recorded.

CLINICAL FINDINGS

Case 1. Baby W was born on 25 February 1954; 'face to pubis' with bruising and oedema of the face and eyes; birth weight 5 lb. 6 oz. There was initial difficulty with feeding and an oesophageal tube was used on 27 and 28 February. Breast feeding was commenced on 1 March, but the baby was lethargic. On this day the mother developed a mild pyrexia; blood films were examined for malarial parasites and found to be negative.

On 4 *March* the baby was noticed to be flushed and hot and its temperature was found to be 100·4°F rising to 103·4° during the night. Its condition became worse towards morning. The skin was pale with some cyanosis, respirations were rapid and the lower extremities were cold. The physical signs on examination were equivocal but the picture generally was suggestive of pneumonia. The white-cell count was 11,400 per c.mm., (neutrophils 46%, lymphocytes 47%, monocytes 4%, immature cells 3%). No malarial parasites were detected. The stool and urine tests gave negative results.

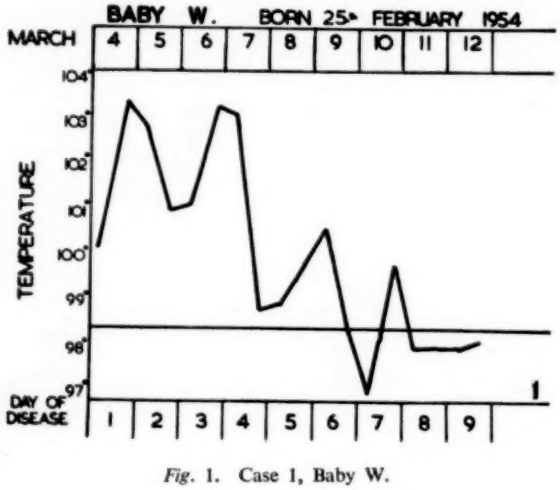

Fig. 1. Case 1, Baby W.

Penicillin (200,000 units) was given 6-hourly, and chloromycetin (75 mg.) 3-hourly. Nasal feeds were given 3-hourly and oxygen administered continuously. This treatment was continued until 6 March with no obvious improvement. There was now slight head retraction but no other signs of cerebral nervous irritation. Aureomycin in 25-mg. doses was substituted for chloromycetin. The baby remained very ill with a raised temperature up to 103° F (Fig. 1).

On 7 *March* there was still no change; the temperature remained elevated and there was considerable retching of mucus. At this stage it was decided to give quinine in spite of negative blood findings and 2½ gr. was given intramuscularly at 8.30 a.m. and repeated at 5.30 p.m. The temperature fell, but there was no corresponding improvement in the baby's general condition.

8 *March.* Quinine was continued, but all other drugs were discontinued. Vomiting was marked.

9 *March.* Baby was still limp and lethargic. Nasal feeds were continued. The temperature returned to normal during the day. The last quinine injection was given at 12 noon.

10 *March.* There was some improvement in the baby's condition and nasal feeding was continued.

11 *March.* The improvement was maintained and feeding by bottle was complemented by tube.

12 *March.* Returned to breast feeding. Continued improvement.

14 *March.* Discharged. Weight 6 lb 5 ozs.

Follow-up. No recurrence of symptoms. Normal gain in weight. No drugs given.

Case 2. Baby T, male, was born on 26 February 1954. Normal delivery; slow to cry; some cyanosis; birth weight 8 lb 8 oz.

Normal progress until 3 *March*, when baby became lethargic and difficult over feeding. Thyroid, gr. 1/10, given thrice daily with apparent improvement.

6 *March.* Baby lethargic and disinclined to feed. Temperature 100·4°F at 8 a.m. No abnormal physical signs on examination. Stools of normal appearance. Penicillin (200,000 units) and aureomycin (25 mg.) given 6-hourly.

7 *March.* Baby still lethargic, pale and ill. Breast feeds taken fairly well. Penicillin and aureomycin continued.

8 *March.* The baby's temperature still elevated and condition unsatisfactory. The white-cell count was 13,400 c.mm., (neutrophils 50%, lymphocytes 44%, monocytes 3%, eosinophils 1%, immature cells 2%.) Urine reaction acid, trace of albumin, no sugar; microscopical examination showed scanty pus-cells, occasional red blood-cells. A specimen of stool was sent to the South African Institute for Medical Research for virus studies.

9, 10 and 11 *March.* The baby's condition was unchanged; he was still lethargic and his temperature still elevated; penicillin was discontinued (Fig. 2).

12 *March.* Vomited mucus in quantity; mouth sore and red.

13 *March.* The vomiting diminished and he was able to breast feed. This was complemented. Temperature normal.

14 *March.* Still lethargic but improvement continued.

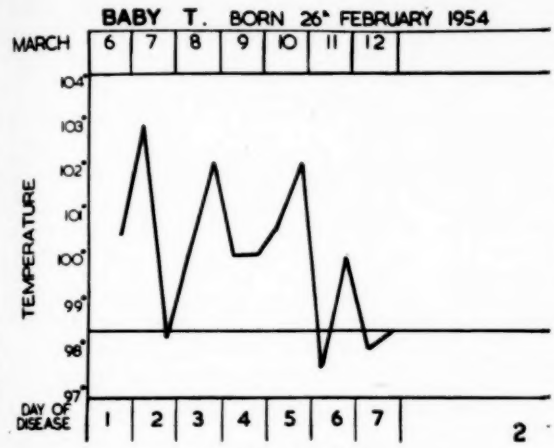

Fig. 2. Case 2, Baby T.

15 *March.* His improvement was maintained, and he was discharged from the Home. Weight 9 lb.

Case 3. Baby B was born on 2 March 1954; forceps delivery, birth weight 6 lb 1 oz.

5 *March.* Baby was lethargic and his attempts to feed were poor. His temperature was raised. Aureomycin (25 mg.) given 6-hourly.

6 *March.* No significant signs were found on physical examination. He vomited and aureomycin was discontinued. Penicillin (200,000 units) 6-hourly given. Nasal feeds were commenced owing to failure to suck. His stools were loose.

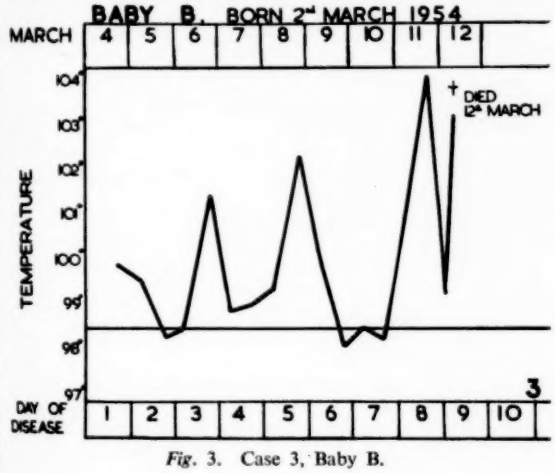
BABY B. BORN 2nd MARCH 1954
MARCH
4 5 6 7 8 9 10 11 12
TEMPERATURE
104° 103° 102° 101° 100° 99° 98° 97°
† DIED 12th MARCH
DAY OF DISEASE
1 2 3 4 5 6 7 8 9 10
3

Fig. 3. Case 3, Baby B.

7 *March.* Respirations and pulse rapid and there was some distension of abdomen. Colour pale and vomiting marked. Temperature remained elevated.

8 *March.* Slight improvement.

11 *March.* Temperature rose sharply, stools loose and offensive (Fig. 3).

12 *March.* Respirations and pulse were fast. Convulsions occurred at 8.45 a.m. Baby died at 9.30 a.m. A post-mortem examination was held at 2 p.m. the same day.

Post-mortem Examination

The external appearances were not significant. The lungs were congested, but there was no bronchopneumonia. The heart showed no congenital abnormalities and macroscopically appeared relatively normal. The abdominal organs showed congestion. The brain appeared normal.

The organs preserved in formol saline and the brain in sterile glycerin were personally taken to Johannesburg on 17 March for further studies. A specimen of stool taken on 11 March was also submitted for virus studies.

PATHOLOGICAL EXAMINATION

In the laboratories of the Poliomyelitis Research Foundation histological sections were prepared from the brain, the heart, one lung, the liver, one kidney and one suprarenal. The results of microscopical examination of these sections were as follows:

Brain. Three sections prepared from the cortex and the midbrain showed congestion but no other pathological change. No foci of inflammation such as were seen in sections of the brain of one of the fatal cases in the previous outbreak of a similar nature in Johannesburg [1, 2, 3] were observed.

Heart. The sections of the heart, which macroscopically had not appeared grossly abnormal, on microscopical examination revealed scattered foci of inflammation in the substance of the muscle. These foci were not circumscribed but faded into relatively normal cardiac muscle. In the foci, the muscle showed eosinophilic degeneration and fragmentation and in places the muscle had disappeared, associated with a surrounding inflammatory cell infiltrate mostly of mononuclear cells, histiocytes and lymphocytes, but also including polymorphonuclear leucocytes.

Lung. The lung showed very marked congestion, but no consolidation or inflammation. The epithelium of the bronchioles was relatively normal and there was no peribronchial inflammation. The pleura was oedematous and there were a few inflammatory cells present.

Liver. The liver showed marked congestion and some of the parenchymal cells showed vacuolation, but there were no foci of degeneration.

Kidney. The kidney sections also showed marked congestion, but no inflammation.

Suprarenal. The suprarenal showed very marked congestion of the medulla, in which a few foci of inflammatory cells mostly of the mononuclear type were detected.

This examination revealed that the only organ showing marked pathological lesions was the heart. The other changes observed, particularly the marked congestion of the lungs and abdominal organs, could have resulted from the failure of the heart, which emerges as the cause of death.

VIRUS STUDIES

Attempts to isolate virus from the specimens of faeces received from each of the 3 babies affected and also from the brain of Baby B were made in the Laboratories of the Poliomyelitis Research Foundation. Recalling that a similar outbreak in Johannesburg in 1952[1] had been found to be associated with an infection with Coxsackie group-B virus,[2] this virus was especially sought. In 2 of the 3 cases it was isolated from the faeces.

Methods

A 10% suspension of the material was prepared in nutrient broth. To this a quarter volume of anaesthetic ether was added and allowed to act for 24-48 hours in a +4°C refrigerator. The suspension was then centrifuged at 3,000 revolutions per minute for 1 hour. The aqueous phase underlying the ether layer was withdrawn and transferred to a wide-mouthed specimen-

bottle, which was then placed in a 37°C incubator for 2 hours to allow the dissolved ether to evaporate. Penicillin (100 units per ml.) and streptomycin (100 μg. per ml.) were added to the suspension, which was then ready for inoculation.

The suspensions prepared from the mouse carcases were treated in a similar manner in some instances, but in others the suspension was not subjected to the action of ether.

Newborn baby mice (less than 24 hours old) were inoculated subcutaneously and then observed for signs of illness, including weakness, paralysis and tremors, for 14 days.

These litters were housed in an isolation unit where no other work was being done with Coxsackie virus and so there was no possibility of cross-infection or of false-positive results.

An account of the relevant details of these studies follows.

BABY W.

13 *March* 1954. A specimen of faeces from Baby W was received.

17 *March*. After preparing a 1:10 suspension in broth as described above the suspension was inoculated into a litter of 7 one-day-old mice. These were observed for 14 days, but none developed signs of illness.

Virus was not isolated from this patient in this one attempt. The attempt was not repeated, as it undoubtedly should have been, in the light of the subsequent findings.

BABY T.

First Isolation from Faeces

13 *March* 1954. A specimen of faeces from this patient was received.

18 *March*. After preparation, a suspension was inoculated into a litter of 7 one-day-old mice.

23 *March*. One mouse was weak. It was sacrificed and a suspension of its carcase passaged the same day into a further litter of 7 mice.

24 *March*. Two mice were paralysed, the rest had severe tremors. One of the paralysed mice was sacrificed and histological sections of its organs were prepared and examined: The fat pad showed necrosis and acute inflammation of some of the lobules; the limb muscles showed small foci of inflammation with eosinophilic degeneration of some segments of muscle, associated with acute inflammatory cell infiltration.

25 *March*. Of the remaining 4 mice, one was found dead, 2 were paralysed and one had tremors.

Second Isolation from Faeces

17 *March* 1954. A second specimen of faeces from Baby T was received. A 10% suspension was prepared and treated as before.

19 *March*. This suspension was inoculated into a litter of 7 one-day-old mice.

20 *March*. One was found dead and discarded.

22 *March*. One had slight tremors.

25 *March*. One was weak. It was sacrificed and its carcase kept for passage.

26 *March*. Three had tremors.

27 *March*. One was paralysed and the rest had tremors. The paralysed mouse was sacrificed and histological sections prepared from its organs: The brain sections showed perivascular cuffing of blood vessels in some areas, pyknosis of the nuclei of some cells, a few inflammatory cells, and small areas of softened white matter. The fat pad showed extensive acute necrosis and inflammation. Lesions shown by each of the 2 mice examined are similar to those produced by Coxsackie group-B virus.

BABY B.

Original Isolation from Faeces

17 *March* 1954. A specimen of faeces collected on 11 March was received. A 10% suspension was prepared in nutrient broth and treated with ether as described above.

19 *March*. The suspension was inoculated into 7 one-day-old mice.

22 *March*. Two of these baby mice appeared to be weak.

23 *March*. Another mouse had developed weakness. One was sacrificed and its organs preserved in Bouin's fixative solution for the preparation of histological sections: Histological section of the brain showed small foci of cells showing pyknosis of the nuclei, associated with a few inflammatory cells. The fat pad showed marked acute necrosis and inflammation. The heart muscle showed foci of eosinophil degeneration and inflammation. These lesions resembled those produced by and characteristic of Coxsackie-B virus infection.

Original Isolation from Contents of Caecum

19 *March*. A suspension prepared from the caecal contents taken *post mortem* was inoculated into a litter of 7 one-day-old mice.

22 *March*. Two of these baby mice were weak. One was kept for passage and a suspension prepared from its carcase was inoculated into another litter of 7 baby mice.

23 *March*. One mouse had wrist drop, 2 were weak and 3 appeared normal.

26 *March*. Three mice were dead, one paralysed and 2 had marked tremors.

Passage 1

22 *March*. The suspension from the carcase of the weak mouse was inoculated into a litter of 7 one-day-old mice.

25 *March*. Five mice were dead; 1 was paralyzed and was sacrificed. Its carcase suspension was kept for further passage. The organs were prepared for histological sections: The brain showed no lesions; the fat pad showed necrosis and early acute inflammation resembling that produced by Coxsackie group-B virus.

Brain. Several attempts were made to isolate virus from a suspension of the brain. Some of these were invalidated by the persistence of post-mortem contaminating bacteria. One gave negative results.

Comment

Viruses resistant to the action of ether and pathogenic to baby mice were thus isolated from the faeces of Baby T on 2 occasions and from the faeces and caecal contents of Baby B. One attempt to isolate virus from the faeces of Baby W failed, as did several attempts to isolate virus from the brain of Baby B.

This failure to isolate virus from the brain was not unexpected as no lesions were seen in the histological sections and, when the baby died, he had been ill for one week, a time which would allow of the development of neutralizing antibodies in the blood.

The viruses isolated from Baby B and Baby T have been passaged several times in baby mice and regularly produce in them an acute necrosis and inflammation of the fat pad. In a proportion of these baby mice, focal lesions of the brain, and also focal necrosis and inflammation of the heart muscle and of segments of the voluntary muscles, have been detected.

As noted above in the individual instances, these lesions resemble those produced by Coxsackie group-B viruses.

IMMUNOLOGICAL STUDIES

Immunological Identification of the Viruses

The viruses isolated from Baby B and Baby T were typed against specific antisera prepared in monkeys against Dalldorf's classical strains of Coxsackie group-B viruses.

Method. In these tests each suspension of mouse carcase was diluted to give a titre of approximately 1,000 L.D./50 per mouse dose of 0·05 c.c. This virus suspension, in 0·5 c.c. amounts, was then mixed with an equal amount of neat serum. The mixture was incubated for 1 hour at 37°C and then the mixture was inoculated into 5 of a litter of 7 newborn mice. The remaining 2 mice were inoculated with virus suspension only, to serve as controls for the others.

Results. The results of the tests were as follows:

Result of Test: Survivors/Total

Test No.	*Coxsackie Group Antisera*	*V i r u s Baby B Faeces*	*Caecal Contents*	*Baby T*	*Interpretation*
1	B1	0/5	0/5	0/5	No protection
	B2	0/5	0/5	0/5	No protection
	B3	0/5	0/5	0/5	No protection
	B4	5/5	5/5	5/5	Protection
2	B1	0/5	0/5	0/5	No protection
	B2	0/5	0/5	0/5	No protection
	B3	0/5	0/5	0/5	No protection
	B4	5/5	4/5	5/5	Protection

The control mice in each experiment developed weakness and paralysis.

Comment. It is apparent from these results that the viruses from Baby B and Baby T were fully neutralized by Coxsackie-B4 antiserum, but not by antisera of Coxsackie types B1, B2 or B3. It was concluded, therefore, that these viruses were Coxsackie group-B type-4 viruses.

Blood specimens were not received from the babies either during the febrile illness or in convalescence. It therefore was not possible to determine whether a rise in titre of their antibodies against Coxsackie group-B virus had occurred subsequent to their infection. The question thus arises as to the pathogenicity of this virus and of its relation to this outbreak of illness.

The same type of Coxsackie group-B virus was isolated from a number of cases of Bornholm disease, which has now been proved to be one manifestation of infection with the virus, and which was prevalent in Southern Rhodesia at this time. During the same period this virus was also isolated from the faeces and the cerebrospinal fluid of a number of cases of meningo-encephalitis. There is therefore little doubt of its pathogenicity, and its presence in the faeces of the babies is presumptive evidence of its aetiological relationship to their illness. It is realized that this was not definitely proved by its isolation from the blood, which was not attempted.

Immunity Tests on the Mothers' Blood Serum

Three months after receiving the specimens from the babies, blood specimens were received from their mothers. This serum was separated and tested on several occasions against the viruses isolated from Baby B and Baby T, and against Dalldorf's B4 strain.

Method. In this test the serum was mixed with a virus suspension containing approximately 1,000 I.D./50 doses of virus. The serum-virus mixture was shaken thoroughly and incubated at 37°C for 1 hour. The mixture was then inoculated subcutaneously into a litter of 5 baby mice. The 2 remaining mice of the litter were then inoculated with the virus suspension only. The mice were observed for 14 days.

Results. The results of these serum protection tests were as follows:

Result of Test: Survivors/Total

Serum (name)	*Test No.*	*V i r u s B.B.*	*B.T.*	*D.B.4*	*Interpretation*
Mrs. B.	1	0/5		0/5	Negative
	2	0/5			Negative
	3	0/5			Negative
Mrs. T.	1		0/5	0/5	Negative
	2		0/5		Negative
Mrs. W.			4/5	4/5	Positive

Comment. The results of these tests reveal that Mrs. B and Mrs. T had no antibodies against the virus isolated from their own babies, but that Mrs. W had antibodies against Baby-T virus and against Dalldorf's B4 strain.

It will be recalled that before her baby became ill Mrs. W had a short febrile illness. It appears probable that this illness was due to Coxsackie-B4 virus infection. Such an interpretation would account for her own illness, and for her baby's illness. Although no virus was isolated, the clinical picture of Baby W's illness was similar to that of the other 2 babies and there is little doubt that Baby W had the same infection. If his mother's infection had preceded by some time her admission to the maternity home, she would have been immune to this infection, and her baby would have shared this immunity.

The finding that both Mrs. B and Mrs. T had no immunity to their baby's viruses is of significance in that their babies would also have no inherited passive immunity. It is unlikely that Coxsackie group-B virus would cause a severe and in one case a fatal illness in babies whose mothers had immunity to this virus. In babies whose mothers have no immunity, it appears from these findings that this virus may on occasion cause a fatal infection as it does in newborn baby mice.

DISCUSSION

The features of this outbreak are essentially similar to those of an outbreak affecting newborn babies in a maternity home in Johannesburg in October 1952. Of 10 babies affected 6 died after an acute febrile illness ending in circulatory collapse.[1] Post-mortem examinations carried out on 3 of them revealed that the cause of death was acute heart failure resulting from a focal but extensive myocarditis.[2] In one case a focus of inflammation in the brain and involvement of the suprarenal were detected, suggesting that the myocarditis was part of a generalized infection.

Coxsackie group-B virus was isolated from the faeces of 2 of the 3 patients who recovered and who were examined for its presence.[3] Baby mice inoculated with suspension prepared from the brain and the heart of 2 of the fatal cases showed lesions of the fat pad and brain, resembling those caused by Coxsackie group-B virus. It was therefore concluded that the outbreak was caused by Coxsackie group-B virus. This virus was typed and found to be a type-3 virus. At the time of this outbreak Bornholm disease was prevalent in Johannes-

burg as it was, later, at the time of the outbreak in Rhodesia, a coincidence which was significant in view of the findings of the virus studies.

The clinical picture and the pathological findings in the two outbreaks were so similar that a similar aetiology was suspected. The findings incriminating Coxsackie group-B virus in the Rhodesian cases is therefore regarded as mutually strengthening the case that this virus was responsible for both outbreaks.

It is noteworthy that the virus isolated from the Rhodesian cases is of a different type from that isolated from the Johannesburg cases. However, the lesions produced in baby mice by these two types of virus are indistinguishable and it may be expected that they could give rise to a similar disease in baby humans. It is believed that these are the first occasions in which evidence incriminating Coxsackie group-B virus as a cause of myocarditis neonatorum has been forthcoming. This evidence, although not absolutely conclusive, is convincing. A further condition—myocarditis of the newborn—has thus been added to those known to result from infection with Coxsackie group-B viruses, which have already been identified as the cause of Bornholm disease and in this region as one of the commonest causes of meningo-encephalitis.

SUMMARY

In late February and early March of 1954, 3 babies developed an acute febrile illness soon after birth in a maternity home in Southern Rhodesia. One of these babies died on the 12th day after birth; the other 2 recovered after being ill about one week.

Histological sections of the organs taken post mortem from the baby who died revealed that the cause of death was an acute focal but extensive myocarditis. Foci of inflammation were also found in the suprarenal and pleura. The other organs showed marked congestion, but no inflammation.

In virus studies, a Coxsackie group-B type-4 virus was isolated from the faeces of one of the patients who recovered, but not (on one attempt) of the other. The same type of Coxsackie-B virus was recovered from the faeces and from the caecal contents of the baby who died.

Immunity studies were not done on the babies' blood, but tests on specimens of blood taken 3 months after the outbreak from the mothers concerned revealed that 2 of them had no antibodies. The mother who had antibodies had a febrile illness while in the maternity home soon before her baby became ill. It seems probable that this illness was caused by the same infection as later affected the babies.

It is concluded that the cause of this outbreak was a Coxsackie group-B type-4 virus. Coxsackie group-B viruses thus far have been incriminated as the cause of pleurodynia or Bornholm disease, meningo-encephalitis and, as a result of the study of this episode and of a similar one in Johannesburg in 1952, of myocarditis in newborn babies, which may end fatally.

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DETERMINATION OF BLOOD VOLUME BY A SIMPLE ACCURATE TECHNIQUE AND ITS APPLICATION IN ASSESSING PATIENTS FOR MAJOR SURGERY

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The routine investigations for the assessment of the patient for surgery include haemoglobin and haematocrit determinations. These express only a percentage concentration and do not give any quantitative evaluation. Owing to this lack of information the blood volume is not taken into account and, provided the haemoglobin is not less than 12 g. (or 80%), no correction of the blood volume is usually made. The replacement of fluid, however, and of blood in particular, is one of the essential factors in reducing the mortality of major surgical procedures.

A further reduction in morbidity and post-operative mortality can be achieved where the blood volume, based on an accurate quantitative determination, is corrected pre-operatively.

In recent surgical literature many reports have emphasized the need for blood-volume estimation; it would seem that the one factor which has limited its general

application has been the lack of a simple accurate technique. Many methods have been described, particularly using the dye technique, but they have been cumbersome and difficult. Usually the syringe has to be weighed before and after the injection, and the dye has had to be made up afresh for each investigation.

In this communication we shall describe a simplified technique which dispenses with these difficulties and which can be used in any institution; a plea will be made for the routine use of blood-volume studies to assess the pre-operative and post-operative needs of surgical patients.

The first recorded attempt to determine the blood volume was made experimentally in 1854 by Welcker, who bled animals to death. Since this method was only applicable experimentally it was replaced by several indirect procedures during the past century. These methods measure (1) the circulating intravascular fluid (plasma), using Congo Red (Griesbach), Neutral Red (Rowntree *et al.*); Brilliant Vital Red Evans dye (Keith *et al.*); Evans Blue (Dawson *et al.*); radio-active ^{131}I (Koster); Dextran (Birch *et al.*); or (2) the total haemoglobin, using carbon monoxide (Sjöstrand); or (3) the red-cell mass by means of radio-active ^{59}Fe (Gibson *et al.*); radio-active ^{32}P (Reid *et al.*); radio-active ^{51}Cr (Gray *et al.*). Of these methods, the estimation of circulating intravascular fluid has been widely adopted and the use of Evans Blue (T-1824) has come to be regarded as most satisfactory. It has several advantages:

1. The dye is non-toxic and is eliminated slowly from the blood stream. The dye has been reported to enter the reticulo-endothelial system and leaves the circulation slowly in comparison with other dyes. It has been estimated that 5-9% of injected dye disappears from the circulation within the 1st hour of injection and that 50-53% has disappeared by the end of 24 hours (Birch *et al.*). This behaviour of the dye is due to the fact that it is firmly bound to the albumin, and is therefore found only in the plasma (Gregerson *et al.*, 1935).
2. It is non-haemolytic.
3. It is uniformly distributed in the blood after 5-6 minutes (Remmer) and it retains its colour on entering the blood stream (Koster).
4. Slight haemolysis does not affect this method (Remmer), especially when using a Beckman DU Spectrophotometer, which measures the optical density selectively at 620 mμ.

It has been stated that the disadvantages of this test are (1) It cannot be repeated at short intervals because of the retention of the dye by the reticulo-endothelial system (Rawson). Remmer, however, says that the results are made even more accurate if the dye injection is repeated after $\frac{1}{2}$ hour to enable blockage of the reticulo-endothelial system to occur, and estimation performed after the double injection.

(2) The patient may become unpleasantly discoloured. Results agreeing closely with those obtained with T-1824 were given by human serum-albumin linked with ^{131}I (Crispell *et al.*). Recently the use of dextraven for measuring blood volume has come into prominence (Birch *et al.*); the results however are at variance with those obtained with the Evans Blue and ^{131}I techniques, possibly owing to the inevitable plasma volume expansion. On the whole, however, the Evans Blue dye dilution method was considered as the best available technique for our purposes.

MATERIAL AND METHOD

1. *The Dye Solution.* Evans Blue dye T-1824, obtained from British Drug House, is dissolved in 0·9% saline in a concentration of 300 mg. %. Ampoules containing 10 ml. of the dye were standardized and sterilized.

2. *Syringes.* Luer-Lok syringes and needles are used. In order to facilitate the injection of the dye into any vein, it was decided to use No. 15 needles only. For collecting blood any needle of suitable size is satisfactory.

All syringes used for injecting the dye were accurately calibrated. About 0·5 cm. from the tip of the piston of a 10-ml. syringe a diamond pencil mark about $\frac{1}{2}$ inch long was made parallel to the circular edge. The syringe was filled with normal saline and pushed forward until the mark on the piston coincided with the 8 ml. mark on the barrel.

By means of accurate weighing, exactly 5 ml. of saline was ejected through an empty No. 15 needle. The starting and end points were carefully marked on the barrel by means of a diamond pencil. For injecting the dye the syringe was (carefully) filled with dye. After all the air had been ejected the piston was pushed in until the mark on the piston coincided with the first mark on the barrel. Exactly 5 ml. of dye are injected by pushing the piston in until the second mark on the barrel coincides with the mark on the piston.

3. *Construction of the Standard Curve.* In order to construct a standard curve, 5 ml. of the dye solution were injected into known volumes of saline, ranging from 1 litre to 5 litres. After thorough mixing the

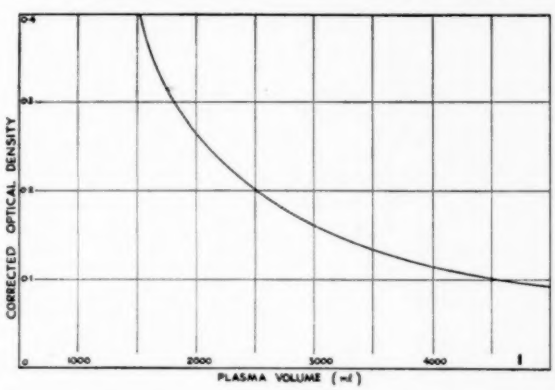

Fig. 1

optical densities were read on a Beckman DU Spectrophotometer at 620 mμ. The data thus obtained were used for the construction of a standard curve (Fig. 1). The curve was found to be a hyperbola which could be represented by the equation:

$$(OD)\,(V)=K. \quad \ldots\ldots (1)$$

where OD=optical density at 620 mμ, V=volume in ml., and K=a constant.

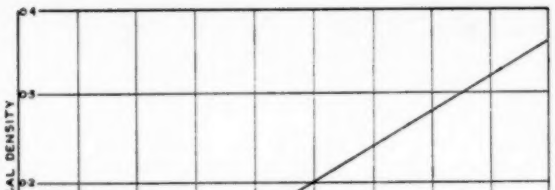

Fig. 2

From (1) $OD = K \div V$ it therefore follows that if the optical density is plotted against the reciprocal of the volume a straight line of slope K will be obtained (Fig. 2). This graph was used in all determinations in preference to that shown in Fig. 1.

4. *Resting Sample and Injection of Dye.* The patient must have fasted for at least 12 hours before the test in order that clear serum may be obtained. Throughout the test the patient has to lie flat in bed; out-patients were rested for 1 hour before the test. A No. 15 needle attached to a syringe was inserted into an antecubital vein, and 12 ml. of blood were withdrawn with the tourniquet released. The syringe was detached, leaving the needle *in situ*. At this stage it will be seen that blood will drip slowly out of the needle, indicating that the needle is well placed in the lumen of the vein.

About 3 ml. of blood are placed into a sedimentation-rate tube for the first haematocrit reading, and thoroughly mixed with the anti-coagulant contained in the tube; the remainder is put into a sterile tube, allowed to clot, and marked 'Resting' sample. A calibrated syringe filled to the upper mark with sterile dye from an ampoule is now securely screwed into the needle. A stop-watch is started as soon as the dye injection is begun. Exactly 5 ml. of the dye solution are injected over a period of 30-40 seconds by pushing the plunger into the barrel until the second mark on the barrel coincides with the mark on the piston.

5. *Samples after Dye Injection.* After 10 minutes 12 ml. of blood are withdrawn into a dry syringe from the other arm with the tourniquet released. About 3 ml. are used for a second haematocrit reading and the remainder is placed into a sterile tube and marked No. 1. The correct time of collection is noted to the nearest quarter-minute.

A last blood specimen is collected 20 minutes after injection, and the exact time is noted to the nearest quarter-minute.

6. *Colorimetry.* The blood samples are spun and the sera transferred to clean dry tubes and centrifuged again. Of each serum 2 ml. are diluted to 5 ml. with 0·9% saline and again centrifuged. The optical densities are read at 620 mμ on a Beckman DU Spectrophotometer.

7. *Calculation.* The optical densities of the coloured solutions are corrected by subtracting the optical density of the resting specimen.

A correction is made for dye disappearance during the tests by plotting the logarithm of the corrected optical densities against time (linear) and determining the extrapolated density value (Ellis). This value (at time= 0 minute) is used in conjunction with Fig. 2 in determining the serum volume. The total blood volume and red-cell volume are calculated from the serum volume and the corrected average haematocrit readings. Haemoglobin determinations are carried out on the two samples collected for haematocrit determinations and an albumin-globulin determination is done on the serum from the resting blood specimen.

It must be remembered that blood is not a homogeneous fluid, but a suspension of corpuscles of varying size in a fluid which is undergoing variation in volume, so that the concentration of corpuscles is not constant throughout the circulation. Methods dependant upon the injection and dilution of a known amount of substance into the blood stream measure only the volume through which the blood carrying the injected substance circulates and it is not possible to differentiate between changes in circulating volume due to fluid loss or to vasomotor or circulatory changes (Tice, editor). It is obvious that, despite all improvements, the dye-dilution method cannot be considered a precise method for measuring or following plasma volume (Courtice and Gunton). It is, nevertheless, useful for comparing values obtained in the same individual at various intervals and is more accurate in this respect than the concentration of plasma protein or the relative volume of packed red-cells.

ILLUSTRATIVE CASES

During the past 2 years blood-volume estimations have been increasingly used in our surgical wards and now it has become largely routine in major surgical cases. Over 80 cases have been fully investigated and a follow-up blood volume done in most of them.

It will be seen that in practically all cases cited the haemoglobin determination, even where corrected by the P.C.V., is misleading. A haemoglobin reading of 13 or 14 g.% does not exclude a red-cell volume of less than 25 ml./kg as seen in cases 1, 2 and 3.

The correction of the blood volume was estimated on the basis of a 3·6 ml./kg red-cell deficiency. (i.e. 10% of red-cell volume) requiring one pint (575 ml.) of whole blood. Where, however, a total of more than 3 pints was required this was given by two separate transfusions. It is better to err on the side of under-estimations, for any further deficiencies can be made up during the operation as required.

Case 1. E.J., female aged 57. Admitted for gastrectomy for gastric ulcer on lesser curvature.

Pre-operative Investigations (9 June 1954):

Weight = 51·4 kg.	Corrected P.C.V. = 41%
Serum total proteins = 7·2 g.%	Total blood volume = 68·1 ml./kg.
Serum albumin = 3·4 g.%	Plasma volume = 40·2 ml./kg.
Serum globulin = 3·8 g.%	Red-cell volume = 27·9 ml./kg.
Haemoglobin = 14·1 g.%	

Correction. Two pints of whole blood were given. A 90% gastrectomy for two high gastric ulcers was performed on 10 June.

Subsequent Investigations (26 July):
Weight = 42 ·6 kg. Corrected P.C.V. = 39%
Total serum protein = 5 ·9 g.% Total blood volume = 66 ·5 ml./kg.
Serum albumin = 3 ·5 g.% Plasma volume = 40 ·5 ml./kg.
Serum globulin = 2 ·4 g.% Red-cell volume = 26 ·0 ml./kg.
Haemoglobin = 15 ·3 g.%

8 September:
Weight = 46 ·4 kg. Corrected P.C.V. = 39%
Total serum protein = 6 ·5 g.% Total blood volume = 57 ·9 ml./kg.
Serum albumin = 4 ·9 g.% Plasma volume = 35 ·2 ml./kg.
Serum globulin = 1 ·6 g.% Red-cell volume = 22 ·7 ml./kg.
Haemoglobin = 14 ·4 g.%

No further correction was done before the patient was discharged.

Case 2. M.B., female aged 58. Admitted for gastrectomy for duodenal ulcer.

Pre-operative Investigations (23 June 1954):
Weight = 46 ·4 kg. Corrected P.C.V. = 40%
Total serum protein = 5 ·7 g.% Total blood volume = 58 ·2 ml./kg.
Serum albumin = 3 ·1 g.% Plasma volume = 34 ·3 ml./kg.
Serum globulin = 2 ·6 g.% Red-cell volume = 23 ·9 ml./kg.
Haemoglobin = 13 ·8 g.%

Correction. Three pints of whole blood were given.

No lesion was found at laparotomy and no further procedure was carried out. This case illustrates an over-enthusiastic correction with some congestion of neck veins which subsided within twelve hours.

The pre-operative correction of anaemia has long been accepted as necessary in skin-graft surgery. Case 3 shows how misleading a picture a haemoglobin determination gives and further illustrates the rapidity of healing after restoring the blood volume to normal limits.

Case 3. M.M., female aged 50. Admitted for a skin graft for an intractable calf ulcer resulting from a burn from a hot-water bottle following a panhysterectomy for carcinoma of the cervix 4 months earlier.

Pre-graft Investigations:
Clinically she appeared well. Haemoglobin = 13 ·3 g.%.
P.C.V. = 38 ·5%.

A routine skin-grafting operation was done. Five days after operation the grafted area revealed a loose slough of skin indicating complete failure of the graft.

Investigations:
Weight = 44 kg. Plasma volume = 34 ·0 ml./kg.
Corrected P.C.V. = 34 ·5% Red-cell volume = 17 ·9 ml./kg.
Total blood volume = 51 ·9 ml./kg.

Correction. 4½ pints of whole blood were transfused in 24 hours without any signs of overloading. Three weeks after the transfusion, the large chronic ulcer was nearly completely epithelialized without further recourse to skin grafting.

There is little doubt in our experience that the pre-operative correction of blood volume is the only accurate means of preparing cases when actual loss of blood has occured. Case 4, a case of long-standing melaena, demonstrates this well. Case 5 (Hodgkin's hypersplenism) also illustrates that one transfusion can rapidly restore haemoglobin levels to normal while red-cell volume is still dangerously low.

Case 4. H.G.H., male aged 67. Admitted with a history of weakness, loss of weight and melaena stools for the past 3 months. There was no history of alcohol.

Pre-operative Investigations (28 May 1954):
Weight = 62 ·5 kg. Total blood volume = 73 ·6 ml./kg.
Haemoglobin = 13 ·8 g.% Plasma volume = 47 ·1 ml./kg.
Corrected P.C.V. = 36% Red-cell volume = 26 ·5 ml./kg.

Barium meal revealed a chronic penetrating duodenal ulcer.

Correction. Two pints of whole blood were transfused.

Subsequent Investigations (3 June):
Weight = 63 kg. Plasma volume = 39 ·2 ml./kg.
Corrected P.C.V. = 42 ·5% Red-cell volume = 29 ·0 ml./kg.
Total blood volume = 68 ·2 ml./kg.

Correction. Two pints more of whole blood were transfused and after this his general condition improved markedly.

A subtotal extensive gastrectomy was performed. The patient made an uninterrupted recovery except for the development of a mild pulmonary atelectasis.

Case 5. E.C., female aged 54. Referred by the physicians for splenectomy because hypersplenism had occured in the course of generalized Hodgkin's disease.

Pre-operative Investigations (7 June 1954):
Weight = 48 ·6 kg. Corrected P.C.V. = 27%
Total serum protein = 5 ·6 g.% Total blood volume = 68 ·7 ml./kg.
Serum albumin = 3 ·5 g.% Plasma volume = 50 ·2 ml./kg.
Serum globulin = 2 ·1 g.% Red-cell volume = 18 ·5 ml./kg.
Haemoglobin = 9 ·6 g.%

Correction. As the plasma volume was already high 2 pints of packed cells were given.

Subsequent Investigations (16 June):
Weight = 47 ·7 kg. Corrected P.C.V. = 37%
Total serum protein = 6 ·0 g.% Total blood volume = 66 ·7 ml./kg.
Serum albumin = 3 ·4 g.% Plasma volume = 42 ·0 ml./kg.
Serum globulin = 2 ·6 g.% Red-cell volume = 24 ·7 ml./kg.
Haemoglobin = 14 ·5 g.%

Correction. As the red-cell volume was still low a further pint of packed cells was given and a transthoracic splenectomy was then performed.

Investigation 1 month after operation (20 July):
Weight = 46 ·6 kg. Corrected P.C.V. = 48%
Total serum protein = 4 ·4 g.% Total blood volume = 79 ·5 ml./kg.
Serum albumin = 3 ·0 g.% Plasma volume = 41 ·3 ml./kg.
Serum globulin = 1 ·4 g.% Red-cell volume = 38 ·2 ml./kg.
Haemoglobin = 16 ·8 g.%

The convalescence was remarkably smooth and on leaving hospital the blood studies were quite normal.

POST-OPERATIVE CORRECTION

These cases illustrate the rapid and marked improvement that takes place after a stormy convalescence when the blood volume is restored to normal.

Case 1. S.S., female aged 62. Eleven years' history of peptic ulcer with repeated haematemeses over the past 6 years. An emergency gastrectomy for uncontrollable bleeding was performed. At the time of operation the patient was in a poor condition. Pathology showed a chronic gastric ulcer. At operation 2½ pints of whole blood were transfused.

Post-operative Investigations:
9 April 1954. Haemoglobin = 13 ·4 g.%. The patient began to have melaena stools.
20 April. Haemoglobin = 15 ·2 g.%.
1 May. A small localized abdominal abscess developed and was drained.
12 May. Five weeks after the operation the patient was still making a very slow recovery.
Weight = 45 ·7 kg. Plasma volume = 25 ·2 ml./kg.
Haemoglobin = 15 ·8 g.% Red-cell volume = 14 ·2 ml./kg.
Corrected P.C.V. = 36% Red-cell count = 5,250,000 per c.mm.
Total blood volume = 39 ·4 ml./kg.
15 May. Two pints of whole blood were transfused.
24 May. Patient had lost a further 3 lb. in weight and therefore another 2 pints of whole blood were given. The patient then began to gain steadily and was soon fit enough for discharge.

Case 2. C.B., male aged 58. A diabetic admitted with a large carbuncle of the neck. He showed very slow improvement on a variety of antibiotics.

Investigations 5 weeks after admission:

Weight = 58 ·2 kg.	Corrected P.C.V. = 40 ·5%
Total serum protein = 8 ·0 g.%	Total blood volume = 53 ·3 ml. /kg.
Serum albumin = 2 ·7 g.%	Plasma volume = 31 ·8 ml. /kg.
Serum globulin = 5 ·3 g.%	Red-cell volume = 21 ·5 ml. /kg.
Haemoglobin = 14 ·4 g.%	

The very slow healing here is partly explained by the low red-cell volume.

Case 3. K.S., male aged 50. An abdomino-perineal excision of the rectum was performed. Post-operatively he had a stormy passage. He had a burst abdomen and a partial retraction of the colostomy, with widespread infection of the abdominal wall. After 4 weeks a large ulcerated area round the colostomy showed little signs of healing.

Investigations (12 May 1954):

Weight = 50 ·0 kg.	Plasma volume = 43 ·5 ml. /kg.
Corrected P.C.V. = 30%	Red-cell volume = 18 ·3 ml. /kg.
Total blood volume = 61 ·8 ml. /kg.	

Correction. 5½ pints of whole blood were given over a period of 10 days with a marked improvement in his general condition and in about 3 weeks he began to gain weight and the ulcerated area was clean and practically healed.

DISCUSSION

Our chief concern has been to show that blood-volume studies are of fundamental importance in assessing the fluid requirements of patients for major operations. We would stress that the haemoglobin and haematocrit values are too frequently misleading to be the sole guide to the quantitative state of the patient's circulating fluid and red-cell mass.

Berlin and his co-workers, using radio-active phosphorus to measure blood volume, give their normal values for males and females as 38 ml. per kg. for plasma and 28 ·5 ml. per kg. for red-cell volume. Ellis's figures for the Witwatersrand (5,740 ft. above sea level), using the Evans Blue method, are 39 ml. per kg. for plasma and 36 ml. per kg. for red-cell volume. At sea level the corresponding figures are 45 ml. and 38 ml. respectively. These blood-volume figures at high altitudes agree closely with those reported by Huey and Holmes at a similar altitude.

In a man of average size a 10% blood volume is equivalent to one pint or 575 ml. of blood. Grant and Reeve have shown that the average blood loss from moderate wounds of skin and muscle implies a 30% loss of blood volume, while very large wounds indicate a 50% blood-volume loss.

Using the described dye technique to determine the plasma and red-cell volumes, and the stated average normal values, the amount of blood to be administered for correction was calculated from the difference between the present blood volume and that calculated for the ideal weight of the patient.

All patients are weighed on admission and the ideal weight is then read from a chart where the calculation is made from height and habitus. In the majority of our patients the ideal weight has been within 10 kg. of the weight on admission. In these cases the ideal weight has been taken as a basis for fluid requirements. In cases where obesity or thinness results in a weight differing widely from the ideal we have in the obese taken the ideal weight, and in the thin the usual weight of the patient. By 'usual weight' is meant the weight over the past few years before the illness. In both cases, therefore, the danger of excess fluid is avoided.

We are investigating further the relation of blood volume to marked overweight and underweight.

Overloading of the circulation and consequent cardiac failure does not appear to be a serious hazard in patients who have proven plasma or red-cell deficiencies. However, in view of the frequent objections raised by anaesthetists that the patient has been or will be 'overloaded' with fluids, it is worth noting that the inevitable loss at operation usually corrects this. It is probably wiser to under-correct by 500 ml. than to over-correct. Frequently 3 or 4 pints of blood have been given pre-operatively without any untoward complications.

During the past 5 years we have noted that the mortality rate in elderly patients undergoing surgery is still high from delayed peripheral circulatory collapse and pulmonary complications. Although care has been taken with the administration of fluids and electrolytes patients die despite all treatment.

Clark *et al.* use the term 'chronic shock' to describe the prolonged peripheral circulatory failure which occur in the post-operative course of those patients who are underweight and who have low blood volumes; they ascribe this to the low reserves of haemoglobin and plasma proteins, which cannot be rapidly mobilized. A few hours' low blood pressure can render important organs, particularly the liver, anoxic; this in turn gives rise to vaso-depressor materials (V.D.M.) which cause irreversible shock (Zweifach *et al.*). Death often occurs in 4-6 days, and at autopsy no macroscopic cause is found.

We prefer the term 'latent shock' to express this concept of chronic shock in the elderly mal-nourished or emaciated patient, because empirical clinical experience shows that poor surgical risks are given a better chance of survival when their haemoglobin and protein reserves are increased by pre-operative blood transfusions.

According to Whipple, when a deficiency of both haemoglobin and tissue protein occurs the formation of new haemoglobin has priority. Consequently the restoration of protein deficits is expedited by the initial correction of a masked anaemia.

We have frequently noted a marked drop in haemoglobin level and fall of the red-cell mass about the 7th post-operative day; this must certainly be one of the main factors in delaying the healing of some wounds and causing wound disruption.

In the post-operative state the great loss of protein as shown by the negative nitrogen balances (Cuthbertson, 1932) and the loss of weight are in need of much closer attention. Post-operative weight-gain must necessarily be slow if there is not enough available protein for the building up of muscle tissue. In the cases noted the rapid improvement and the gain in weight following the correction of blood volumes, especially after a stormy convalescence, is testimony to the efficacy of accurate replacement.

SUMMARY AND CONCLUSIONS

A simple accurate technique for the determination of blood volume using the Evans Blue dye method is described.

A series of cases is presented showing the fundamental values of blood-volume studies in the pre-operative assessment of the patient, and in the reduction of post-operative morbidity.

We are indebted to Dr. H. B. W. Greig of the Department of Haematology at the South African Institute for Medical Research for the P.C.V. and haemoglobin estimations, and to Mr. C. Solomons for technical assistance and mathematical calculations. We also should like to thank Dr. K. Mills and Prof. W. E. Underwood of the Medical School of the Witwatersrand University, and the Director of the South African Institute for Medical Research, for their permission to carry out this research and publish this paper.

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ENDEMIC GOITRE IN SOUTH AFRICA

The Union Department of Nutrition has published a monograph* serving *inter alia* as a report of the investigations of the Goitre Research Committee appointed 6 years ago under the auspices of the Department of Health, the University of Pretoria, and the Council for Scientific and Industrial Research.

The researches of the Goitre Research Committee are described, the results summarized, and the recommendations made to the Union Government set out. In addition the subject of goitre is treated on general lines (classification of thyroid enlargements, causes of simple or endemic goitre, the symptoms of endemic goitre, prophylaxis etc.).

The Committee's method of approach was to make enquiries concerning goitrous, suspected goitrous and non-goitrous areas through the Departments of Education of the 4 Provinces, the Union Department of Education, and the Department of Health. Schools in the areas concerned were visited and the children examined. Subsequently the homes of the children found to be suffering from goitre were visited and the remaining members of their families examined, and specimens of food and drinking-water collected for analysis.

For purposes of comparison the homes of children free from goitre were visited and similarly investigated.

As many areas as possible were visited, and in addition many private practitioners, district surgeons, medical inspectors of schools, and others, were contacted, who submitted specimens of food, soil and water from goitrous and non-goitrous areas for analysis.

Endemic goitre was reported from the following areas:

UNION OF SOUTH AFRICA

Cape Province

Langkloof-Cougha River area (near Port Elizabeth). The whole of this area can be considered an endemic goitre area, the worst parts being Noeree River valley and the Opkoms area. The number of goitre cases (including slight thyroid enlargements) varied from 3% to 22% in the different schools. A fair number of adults, mostly females, have been operated on for toxic goitre at Port Elizabeth. The whole area (including the Knysna-George area) is known to be deficient in phosphorus and calcium, which is reflected in the very poor teeth of most of the children and adults. In general the diet is fairly satisfactory, containing meat, vegetables and fruit. Fourteen soil specimens in the goitrous area varied in iodine content from 190 μg. to 3,670 μg. per kg., as compared with a non-goitrous Pretoria area 1,500 μg. to 7,500 μg. The drinking-water in the Cougha Mountains is deficient in iodine (6-11 μg. p.l.), and at Braam River (stream water) 22 μg. p.l.

North Western Cape Province. Goitre is endemic in these areas, and it was found that this was not due to iodine deficiency (the drinking-waters are rich in iodine), but to the presence of harmful quantities of fluorine in the drinking-water. The region is well known as an endemic fluorosis area, where teeth are badly mottled. A fair proportion of people, especially women, settling in this part of the country develop thyroid enlargement within 10-15 years. At 20 schools in the places named below 7-25% of the children examined showed evidence of goitre. The iodine and the fluorine in the drinking-waters were as follows:

Place	*Source of Drinking Water*	*Iodine Content (μg. per litre)* *	*Fluorine Content (mg. per litre)* *
Kenhardt ..	River (Municipal supply)	220	*Nil*
Upington ..	Orange River (Municipal)	15	0·05
Kakamas ..	Orange River (Municipal)	15	0·05
Pofadder ..	Boreholes	43-322	2·0-2·6
Springbok ..	Boreholes (Municipal)	50	1·6
O'Kiep ..	Boreholes	42-175	1·7-2·6
Nababeep ..	Boreholes	42-175	1·7-2·6
Port Nolloth	Wells	290-320	*nil*-0·1
Sultana Oord	Orange River ..	15	0·05
Karos ..	Orange River ..	15	0·05
Noenieput ..	Boreholes	270-380	1·4-3·2

* Steyn, D. G., Kieser, J., Odendaal, W. A., Malherbe, H., Snyman, H. W., Sunkel, W., Naude, C. P., Klintworth, H. and Fisher, E. (1955): *Endemic Goitre in the Union of South Africa and Some Neighbouring Territories.* Union of South Africa, Department of Nutrition. Pp. 90, with map and 30 photographs.

Place	Source of Drinking Water	Iodine Content (μg. per litre)*	Fluorine Content (mg. per litre)*
Askham ..	Borehole	22	*Nil*
van Zylsrus..	Boreholes	25-36	*Nil*
Seodin ..	Boreholes	10-14	*Nil*
Kuruman ..	Spring (Municipal) ..	8	*Nil*
Vryburg ..	Boreholes	64	0·3

* One milligram per litre (1 mg. p.l.)=1 part per million. One milligram (mg.)=1,000 micrograms (μg.).

In these areas 200 adults and 4,813 school children were examined. Several men and women stated that with large doses of iodine (usually Lugol's solution) their goitres disappeared. The investigators gained the impression that in areas where the goitre is fluorine-induced the incidence of nodular goitre in adolescents is greater than where the goitre is due to a primary iodine deficiency; also that the treatment of simple goitre with large doses of iodine in the form of Lugol's solution is, to a certain measure at least, responsible for the high incidence of thyrotoxicosis which they found in the area. (The Kenhardt water supply has been in use for a few years only; formerly the municipal supply was from boreholes. The fairly high percentage of goitre in the children at the Kenhardt schools is attributed to (1) the drinking of the previous borehole supply until some years ago, and (2) the fact that a large percentage of the school children hail from farms where the water contains harmful quantities of fluorine.) Generally speaking the diet of the people in this region is satisfactory; a large proportion of the fruit and vegetables is imported.

Transvaal

Bronkhorstspruit area. Cases of endemic goitre in Europeans and Bantu. The iodine in drinking-water from goitrous families varied from undetectable quantities to 5 μg. p.l.

Belfast-Machadodorp area. Many cases in Bantu.

Nelspruit-Barberton area. Prevalent among Bantu (in one district Dr. T. Blake had found 290 cases in about 300 Bantu women). The iodine in drinking-water varied from undetectable quantities to 13 μg. p.l.

Brits area. A small number of slightly enlarged thyroids found in a primary school at Skeerpoort and a lesser degree at a primary school at Wolhuterskop. A few adolescent girls had markedly enlarged glands and 2 women had exophthalmic goitre.

Koster area. Only one of the 8 families living on the farm Kosterfontein was free from goitre; this was the only family who drank borehole water (16 μg. iodine p.l.); the remainder used water from springs flowing from the 'Witwatersrand' (5-10 μg. iodine p.l.). In the other families 2 elderly women had exophthalmic goitre and 3 more had been operated on for the same complaint. The municipal water supply at Koster, where goitre is not known to occur, contains 32 μg. iodine p.l.

Natal

Polela area. An endemic area. The iodine content in 15 specimens of water ranged from undetectable quantities—less than 3 μg. p.l.—(in 5 specimens) to 64 μg. p.l., and the fluorine content from 0·3 to 214 mg. p.l.

Orange Free State

It appears that the entire Drakensberg area, including the whole of Basutoland, can be regarded as an endemic goitre area.

ADJOINING TERRITORIES

Basutoland

See preceding paragraph.

Caprivi Strip

The Caprivi Strip is about 200 miles in length and varies from 20 to 30 miles in breadth; it has a population of about 15,000. It is a portion of South West Africa and is bounded by Angola, Northern Rhodesia, Southern Rhodesia and the Bechuanaland Protectorate. It is fertile and excellent cattle country and the food position of the Natives (who in parts eat a great deal of river fish) appears to be very satisfactory, and their nutritional state is good. The cattle show no signs of goitre. Throughout the area hygenic conditions are very bad.

Eastern Caprivi Strip. This area has been reported as highly goitrous by Major Trollope, Dr. S. Annecke, Dr. B. Squires and others. This is confirmed by the Committee, who conducted investigations amongst the Natives in the villages of Ikoma, Matanga, Linyanti (and its sub-villages), Kanono, Lisikili, Kalaluka and Silitene. They examined 460 persons of all ages. The average incidence of goitre was 50%, and the figures reached 70% at Linyanti. In females between 20 and 40 years old the incidence of nodular goitre was 16-40%. Earlier observers have reported that even breast-fed babies have enlarged thyroids. The drinking-water is obtained from the Chobe and Zambesi Rivers and from the pans and wells in the rest of the country, of which large tracts are inundated during the rainy season. The different waters varied in iodine content from 8 to 36 μg. p.l. The Committee conclude that the high incidence of endemic goitre in the Eastern Caprivi Strip is due almost entirely to a pronounced primary deficiency of iodine in the soil, food and water. As a general rule, the incidence bears a direct relationship to the degree of iodine deficiency in the drinking-water.

Western Caprivi Strip and South West Africa. Dr. W. H. G. Kuschke (1952) surveyed this portion of the Caprivi Strip and reported as follows: 'It appears quite definite from the results .... that goitre is prevalent amongst all these Native tribes, viz. Barakwengo 69·9%, Okavango Natives 62·6% (Mbukush and Derico). The cause .... must be sought in an iodine deficiency in the food, water and soil of these areas. Of all the enlarged thyroids examined, only a few were nodular ....' Dr. Kuschke suggested that iodized salt should be supplied to the inhabitants of the Caprivi Strip and to all the tribes of the Okavango territory.

It therefore appears that the entire Caprivi Strip is an endemic goitre area.

In the semi-arid southern region of the South West Africa adjoining the North-Western Cape Province subterranean waters also contain fluorine.

Swaziland

Drew, Elder and Glynn (1940) called attention to goitre in Swaziland and stated there was a general iodine deficiency there. F. W. Fox recommended the use of iodized salt. The Committee examined 2,000 Native and Coloured school children and 300 European. The percentage of goitrous non-European children in individual schools varied from 4% to 71%. The average amongst Native school children was 26% and amongst the European children 4%. There was a definite tendency for the incidence of goitre to be directly proportional to the degree of iodine deficiency in the water. The Bremersdorp municipal water contained 18 μg. of iodine p.l. and other river supplies 18-22; spring water contained iodine varying from 8 to 59 μg. p.l. The nutritional state of the children was fair and their teeth good.

IODINE AND FLUORINE CONTENTS

A comparison is made between the goitrous regions of Swaziland, Caprivi Strip and Langkloof-Cougha River and the non-goitrous regions of the Colesberg, Middelburg-Cradock area and Griqualand West. The iodine contents are expressed as μg. p.l. or kg. and the fluorine as mg. p.l.

In these goitrous regions the iodine in the soil varied from 190 to 5,400 and in the grass from 900 to 3,500; in the non-goitrous regions the figures were—soil 1,500-7,500, grass 1,500-3,500 (the analyses of grass were made from the point of view of animal nutrition—see below).

In the goitrous regions the iodine in the water varied from less than 5 to 33 (except in regions where the endemic goitre was considered to be due to fluorine, in which the iodine in the water varied from 39 to 820); in the non-goitrous regions the iodine content of the water varied from 39 to 320.

The fluorine content of the water in regions of fluorine-induced goitre varied from 1·4 to 5·5, and in non-goitrous regions from less than 0·05 to 1·2.

Little evidence of goitre in animals was discovered, even in areas where human goitre is endemic. The Committee's investigations indicate that this is because the grasses eaten by stock in these areas contain much larger quantities of iodine than the vegetables, cereals and fruit grown there.

RECOMMENDATIONS OF THE COMMITTEE

Two years ago the Committee recommended to the Minister of Health that iodized salt should be supplied to all endemic goitrous

areas in the Union and to the entire Caprivi Strip and Swaziland, *but not to the areas of fluorine-induced endemic goitre*. The recommendation is now extended to Basutoland and those northern parts of South West Africa where goitre is endemic. The level of iodization suggested was 1 part of iodine per 100,000 parts of salt, and it was recommended that the iodine should be added in the form of potassium iodate (KIO_3) instead of potassium or sodium iodide, as the iodate is much more stable than the iodides. The advice has been accepted by the Government and it is hoped that iodized salt will be available for distribution within the next few months.

The Committee recommends that individuals showing symptoms of thyroid hyperfunction should not take iodized salt except under medical advice.

The use of salt containing 1 : 100,000 parts of iodine is not sufficient to cure existing cases of simple goitre, and it is recommended that these should be supplied with additional iodine.

It is recommended that when the iodized salt is used care should be taken concerning the simultaneous use of medicines or cosmetics containing iodine. A warning is also issued concerning the use of iodides and iodates as food preservatives. The Committee recommends that all preparations containing iodine should carry labels indicating the quantity present.

Special reference is made to the low iodine content of the Pretoria municipal water supply (average 18 μg. p.l.) and parts of the Witwatersrand and Vereeniging, where a few specimens of water drawn from houses contained an average of 23 μg. of iodine p.l. It is recommended that annual surveys be made of school children. in these areas.

The Report includes a map (Fig. 1), from which (the Committee states) 'it is clear that a goitre belt runs from the Letaba area, in the Transvaal, through Swaziland, Natal, the Eastern Cape Province, Southern Cape Province, Villiersdorp, Caledon, Hex River Valley, to Ceres and Tulbagh in the Western Cape Province. It is not maintained that all the inhabitants of this goitre belt are affected by the disease, but that a large number of endemic goitre areas are situated in it. *More intensive and extensive research into our goitre problem will undoubtedly reveal many more endemic goitre areas, not only in the goitre belt but also in other parts of South Africa*'.

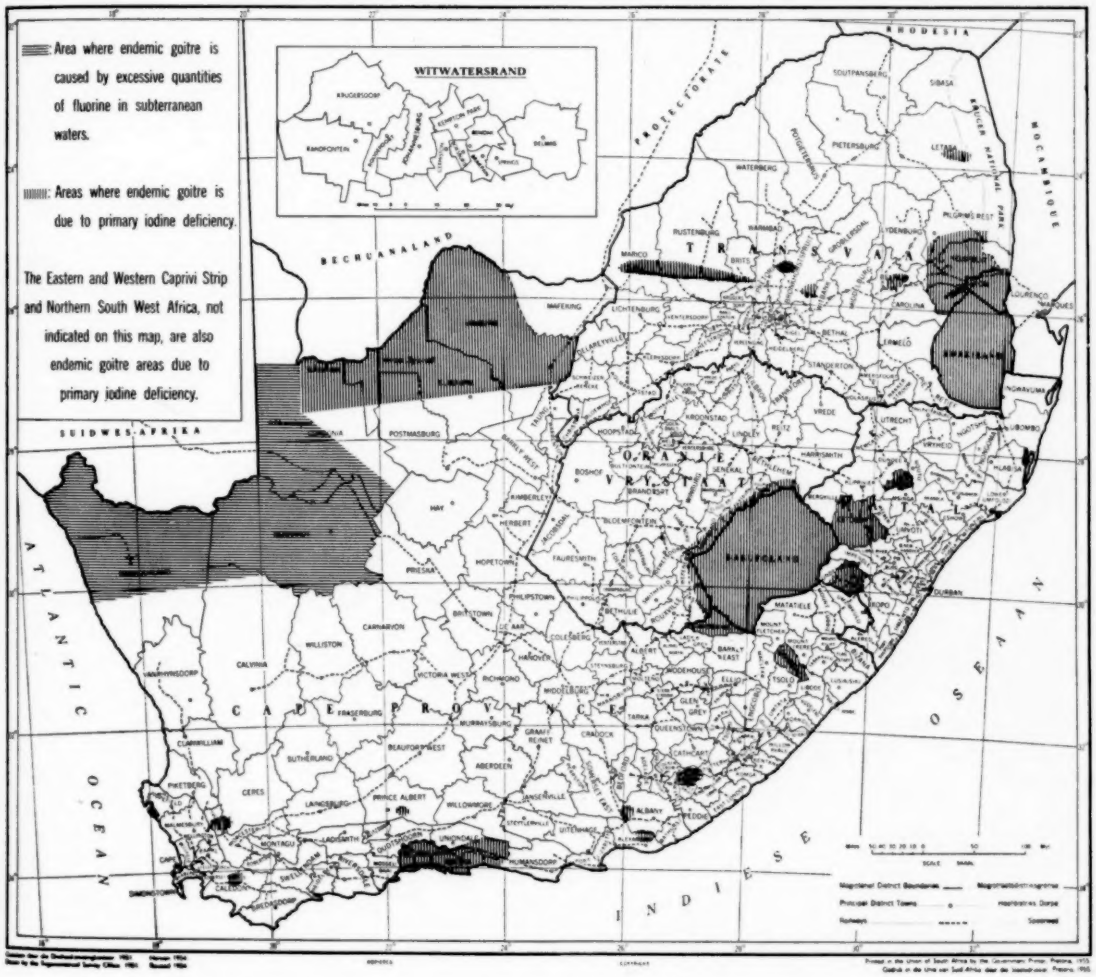
Area where endemic goitre is caused by excessive quantities of fluorine in subterranean waters.
Areas where endemic goitre is due to primary iodine deficiency.
The Eastern and Western Caprivi Strip and Northern South West Africa, not indicated on this map, are also endemic goitre areas due to primary iodine deficiency.
WITWATERSRAND
RHODESIA
BECHUANALAND
SUIDWES-AFRIKA
MOCAMBIQUE
TRANSVAAL
ORANJE VRYSTAAT
NATAL
CAPE PROVINCE
ATLANTIC OCEAN
INDIAN OCEAN

Fig. 1 Map of the Union of South Africa indicating where Endemic Goitre occurs.

WORKMEN'S COMPENSATION FEES : SOUTHERN RHODESIA

The Southern Rhodesian Government has, after consultation with the Mashonaland and Matabeland Branches of the British Medical Association prescribed the following scale of fees for medical aid to workmen (Southern Rhodesia Government Notice No. 169 of 1955).

Visits and Examinations: Per visit, 15s.

Consultations: One doctor with another, £3 3s.

Assistance at Major Operations: 10% of the surgeon's scheduled fee with a minimum of £3 3s.

Simple Wounds: (*a*) Without suture of wound, £1 1s. per attendance, with maximum of £3 3s. (*b*) With suture of wound, £3 3s.

Suture of Tendons or Nerves: (*a*) (i) Suture of single tendon (simple), £12 12s. Each additional tendon, £3 3s. (ii) Repair of flexor tendon of fingers and tendo achilles, £26 5s. (*b*) (i) Suture of single nerve, £18 18s. Each additional nerve, £5 5s. (ii) Transplantation or extensive dissection to mobilize nerve, £26 5s.

Treatment of Abscesses: Superficial abscess, £1 1s. per visit, with a maximum of £4 4s.

Treatment of Infections of Hand, etc.: (*a*) Pulp space infection, £6 6s. (*b*) Suppuration of tendon sheaths, or (*c*) in thenar, midpalmer or forearm spaces, primary or secondary, £12 12s. (*d*) Consequent deep forearm suppuration, £23 12s. 6d.

Removal of Bursa, £4 4s.

Removal of Foreign Bodies (other than from eye): (*a*) Superficial, £3 3s. (*b*) Deep (excluding intraperitoneal), £12 12s.

Fractures:

1. (*a*) Bones of hand and foot (except os calcis, talus and carpal scaphoid), £4 4s. (*b*) Os calcis and talus, £15 15s. (*c*) Non-operative treatment of patella or of carpal scaphoid, £8 8s. (*d*) Upper arm, forearm, leg (one bone), £15 15s. Forearm or leg (both bones), £18 18s. (*e*) Femur, £28 7s. (*f*) Rib or ribs (uncomplicated), £3 3s. (*g*) Spine: (i) Transverse process or spinous process, £10 10s. (ii) Body of vertebra, £31 10s. (*h*) Pelvis: Uncomplicated, £12 12s. Complicated, £39 7s. 6d. (*i*) Clavicle, £6 6s. (*j*) Scapula, £6 6s. (*k*) Jaws: Upper jaw, £21. Lower jaw, £21.

2. Where the fracture is compound and/or complicated the above fees may be increased by 50% except for (1) (*g*) (ii) above.

3. Open operation for treatment of fracture and its complications including operations for mal-union, £23 12s. 6d.

Where an open operation is necessary in addition to any of the other treatments mentioned in this item the fee therefor shall be paid in addition to any other fee which may be payable in terms of this item.

Head Injuries: (*a*) Trephining, £31 10s. (*b*) Osteoplastic operation, £47 5s. (*c*) Lumbar puncture, £2 2s. (*d*) Cerebro-concussion 15s. per visit, with maximum of £15 15s.

Dislocations:

(1) Simple: (*a*) Bones of hand, foot and lower jaw, £4 4s. (*b*) Patella, £5 5s. (*c*) Shoulder, elbow, wrist, ankle, £9 9s. (*d*) Knee or hip, £18 18s. (*e*) Spine, £39 7s. 6d.

(2) Compound: (*a*) Bones of hand or foot, £18 18s. (*b*) All other, £28 7s.

Joint Injuries:

(1) (*a*) Sprains (other than ankle or knee), £2 2s. (*b*) Sprained ankle or knee, £4 4s. (*c*) Penetrating wounds of large joints, £18 18s. (*d*) Operation for internal derangement of knee joint, £31 10s. (*e*) Aspiration of joints, £2 2s. (*f*) Mobilization of joints under anaesthetic, £4 14s. 6d. (*g*) Simple manipulation of spine, £4 14s. 6d.

(2) (*a*) Conservative treatment of prolapsed intervertebral disc requiring P.O.P. Jacket manipulation under anaesthetic, etc., £15 15s. (*b*) Operative treatment for prolapsed intervertebral disc, laminectomy or spinal fusion (the fee to include all forms of preliminary investigation during the preceding 3 months), £42.

Amputations: (*a*) Finger or toe, £4 4s. (*b*) Forearm or below knee, £23 12s. 6d. (*c*) Above elbow or knee, £28 7s. (*d*) Hand, partial, £23 12s. 6d. (*e*) Through shoulder or hip joints, £39 7s. 6d.

Paracentesis of chest, £2 2s.

Intra-abdominal operation, £39 7s. 6d.

Intrathoracic operations: (*a*) For empyema of chest, £15 15s. (*b*) All other, £39 7s. 6d.

(*a*) *Repair of hernia* (single), £31 10s. (*b*) (double), £47 5s.

Injuries to the urinary tract. Operation for ruptured kidney, ruptured bladder, ruptured urethra or ureter, £39 7s. 6d.

Eye:

(*a*) Examination, per visit, including ordinary treatment: First visit, £2 2s. Subsequent visits (per visit), £1 1s. with maximum of £18 18s.

When a subsequent visit includes any of the following specialized examinations the fee for that visit shall be £2 2s.: (i) Complete refraction. (ii) Slip lamp examination. (iii) Visual fields on perimeter and/or Bjerrum's screen. (iv) Examination on Hess chart. (v) Orthoptic examination.

(*b*) Treatment for corneal ulcer or for superficial foreign body, as above. (*c*) Operation for traumatic cataract, £34 13s. (*d*) Intraocular magnet operation, £34 13s. (*e*) Needling, £9 9s. (*f*) Iridectomy, £23 12s. 6d. (*g*) Enucleation, £23 12s. 6d. (*h*) Plastic operations, £18 18s. (*i*) Detachment of retina, £39 7s. 6d. (*j*) Evisceration of eye, £15 15s. (*k*) Suture of conjunctiva, £7 17s. 6d. (*l*) Suture of corneal wounds, £15 15s. (*m*) Paracentesis, £7 17s. 6d.

Ear, Nose and Throat:

(*a*) Examination, per visit, including ordinary treatment: First visit, £2 2s. Subsequent visits, per visit, £1 1s. with maximum of £18 18s.

(*b*) Mastoiditis—Simple, £34 13s. Radical, £39 7s. 6d. (*c*) Paracentesis of one ear, £6 6s. Two ears, £9 9s. (*d*) (i) Simple fracture of nasal bones, £10 10s. (ii) Plastic operation refracture and reduction, £15 15s. (*e*) Intranasal dacryocystotomy, £34 13s. 6d. (*f*) (i) Other minor intranasal operations, e.g., turbinectomy, £10 10s. (ii) Other major intranasal operations, e.g., S.M.R., £18 18s. (*g*) Radical maxillary antrum, £28 7s. 6d. (*h*) External frontal or ethmoid sinus radical operation, £39 7s. 6d. (*i*) Tracheotomy, £18 18s. (*j*) (i) Oesophagoscopy for removal of foreign body, £26 5s. (ii) With dilation of stricture, £21. Additional dilations, £10. (*k*) Bronchoscopy for removal of foreign body, £26 5s. (*l*) Removal of foreign body from ear or nose under anaesthetic, £6 6s. (*m*) Andeometric examination and assessment of percentage hearing loss, £3 3s. (*n*) Labyrinthic tests, £2 2s. (*o*) Antrum puncture, £2 2s.

Radiological:

(1) X-ray examination, including screening where necessary: (*a*) Screen examination only, £3 3s. (*b*) Hand, wrist, forearm and ankle, £3 3s. (*c*) Shoulder, foot, knee, £3 3s. (*d*) Any long bone, £3 3s. (*e*) Urinary tract, plain, £3 3s. (*f*) Urinary tract with uro-selectan, £5 5s. (Fee to cover one injection and four plates. Where those figures are exceeded, £1 1s. additional for each injection after the first and £1 1s. for the fifth and each further plate.) (*g*) Skull (all types), £5 5s. (*h*) Antra, facial and nasal bones, £3 3s. (*i*) Pelvis, £3 3s. (*j*) Chest and ribs for fracture, £3 3s. (*k*) One section of spine, £3 3s. (*l*) Whole spine, £6 6s. (*m*) Gall bladder (plain), £3 3s. (*n*) Gall bladder with dye, £6 6s. (*o*) Stomach with barium meal, £5 5s. (*p*) Gastro-intestinal tract with barium meal, £7 17s. 6d. (*q*) Gastro-intestinal tract with barium meal and gall bladder with dye, £10 10s. (*r*) Stomach only with barium meal plus gall bladder with dye, £8 8s. (*s*) Barium enema, £6 6s. (*t*) Eye-localization of foreign bodies, £5 5s.

(2) Portable X-ray examination away from consulting rooms (additional to above fees), £4 4s.

(3) Where as the result of any one accident X-ray examinations of different parts of the body are necessary the highest prescribed fee for any examination of one part of the body shall be paid in full and the prescribed fees in respect of any other examination shall be reduced by 50%.

(4) Where as the result of any one accident more than one examination is necessary, the examinations subsequent to the first, when undertaken by the same practitioner, radiologist, or hospital within a period of 3 months of the first examination in respect of such accident, shall be charged for at the rate of 50% of the prescribed fee.

Anaesthetics:

(1) (*a*) For operations for which the fee prescribed in these regulations is £12 12s. or less, £2 2s. For operations the prescribed fee for which exceeds £12 12s., £2 2s. plus 10s. 6d. for each £5 5s. or part thereof of the total surgical fee prescribed.

(*b*) Where the anaesthetist supplies his own anaesthetic, the cost of such anaesthetic may be added to the above fee. *Note.*—These fees shall apply to whatever type of anaesthetic has been decided upon, i.e., intravenous, spinal, intranasal or splanchnic and include pre-operative examinations and post-operative visits by an anaesthetist.

(*c*) For local anaesthesia: (*i*) For operations where the prescribed fee is £3 3s. or less, Nil. (ii) For operations where the prescribed fee is more than £3 3s. but not more than £18 18s., £3 3s. (iii) For operations where the prescribed fee is £18 18s. or more, add 10s. 6d. for each £5 5s. or part of same by which the prescribed fee exceeds £18 18s.

Blood Transfusions: For first transfusion of one or two pints (and including grouping and cross matching), £3 3s. For each additional transfusion, £1 1s. with a maximum fee of £10 10s. for any one accident.

Physiotherapy by Registered Medical Practitioners: (*a*) Superficial therapy, £1 1s. per visit (maximum, £21). (*b*) Massage and electrical treatment, £1 1s. per visit (maximum, £21). (*c*) Electrical reactions of muscles, £2. (*d*) All special forms of treatment, £2 2s. for first visit and £1 1s. for each subsequent visit with a maximum of £12 12s.

Scheduled Industrial Diseases: Dermatitis—examination and full report, including patch testing, £8 8s.

Medicines and Dressings: Where drugs and dressings are necessarily and actually supplied by the attending practitioner, 10% may be added to the standard retail tariff of the Pharmaceutical Society of Southern Rhodesia.

Vaccine or Serum: Where in the course of treatment for any injury, it is necessary to administer a vaccine or serum the net cost thereof may be added to any fee payable under the schedule.

Travelling Expenses: A fee of 2s. per mile travelled shall be allowed to a medical practitioner called upon to travel to a place more than 3 miles from his surgery or usual place of residence. No fee shall be payable where the place visited is not more than 3 miles from his surgery or usual place of residence. No detention fee shall be payable in respect of such journeys. Where more than one patient is visited at different addresses on the same journey only the actual miles travelled to visit each patient shall be payable.

NOTES

Where a fee for any service is prescribed herein, the medical practitioner shall not be entitled to payment calculated on a basis of visits or examinations made, where such calculation would result in the prescribed fee being exceeded.

Fees for services not specified in this tariff and fees for specially complicated or prolonged treatment may be referred to the Commissioner for assessment by Medical Assessors to be appointed by the Minister after consultation with the Medical Association of Southern Rhodesia.

For the purpose of this Schedule 'major operation' shall mean any operation for which the fee prescribed is £12 12s. or more.

The cost of all necessary attention subsequent to any operation and/or treatment in respect of which a fee is prescribed in this Schedule, shall be regarded as being included in such fee.

The prescribed fee shall cover the cost of routine reports giving details of an injured workman's absence from duty, the nature of the injury or disease from which he is suffering and the date on which he will be fit to return to duty. For any other report called for by the commissioner, a fee of £2 2s. may be claimed.

Where, as a result of any one accident, more than one operation or treatment for which a special fee in respect of each is prescribed in this Schedule is performed or carried out at the same time by the same medical practitioner, the prescribed fee in respect of any one operation or treatment shall be paid in full and the other prescribed fees shall be reduced by 50%.

Where secondary operations are performed at a date subsequent to a primary operation 50% of the prescribed fee shall be payable in respect of such secondary operations.

The fees and charges in respect of services rendered to Native workmen shall be 50% of the fees herein prescribed.

POLIOMYELITIS IN THE UNION

Following are the returns, supplied by the Union Department of Health, of cases notified under the Public Health Act as suffering from Poliomyelitis in the period 26 May to 2 June 1955.

	European	*Non-European*
Cape Province:		
Fort Beaufort Divisional Council ..		1
Hanover Divisional Council		1
Port Elizabeth Municipality		1
Cape Divisional Council	1	1
Montagu Municipality		1
Total for Cape Province	1	5
Transvaal:		
Alberton Municipality	2	
Greylingstad		1
Germiston Municipality		1
Total for Transvaal	2	2
TOTAL FOR THE UNION	**3**	**7**

Report for the period 3 to 10 June 1955.

Cape Province:		
Beaufort West Divisional Council ..		1
Prieska Municipality	2	
Flagstaff District		1
Total for Cape Province	2	2
Transvaal:		
Louis Trichardt District		1
Total for Transvaal		1
TOTAL FOR THE UNION	**2**	**3**

Union Department of Health Bulletin. Report for the 7 days ended 9 June 1955.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in other Countries:

Plague: Nil.

Cholera in Chalna, Chittagong, Dacca (Pakistan); Calcutta (India).

Smallpox in Kabul (Afghanistan); Phnom-Penh (Cambodia); Allahabad, Bombay, Calcutta, Cochin, Delhi, Kanpur, Kozhikode, Lucknow, Madras, Tellicherry, Visakhapatnam (India); Dacca, Karachi, Lahore (Pakistan); Hué, Nhatrang, Saigon-Cholon, Tourane (Viêt-Nam); Mogadiscio (Somalia).

Typhus Fever in Baghdad (Iraq); Alexandria, Cairo (Egypt).

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Werner Weinberg, Johannesburg, who was previously elected Associate Member of the International Fertility Association, has now been promoted to Active Membership of the same organization. At the same time he has been elected Associate Editor for Abstracts in South Africa of the new international journal *Fertility*, which will have its first issue in July 1955.

* * *

University of the Witwatersrand Medical Graduate Association. A week-end postgraduate course in Surgery has been arranged by the Department of Surgery to take place on 12, 13 and 14 August. Provisional programme (subject to alteration):

Friday 12 *August*

9—12 a.m. Ward Rounds.
2—5 p.m. 1. Diagnosis and Management of Rectal Bleeding.
2. Late sequelae of Surgery of Peptic Ulcer.
3. Surgery of the Gall Bladder and the Appendix.
8 p.m. Symposium on the Diagnosis of the Acute Abdomen in the Child, Adult and Aged.

Saturday 13 *August*

9—12 a.m. Ward Rounds.
2—5 p.m. Common Orthopaedic Conditions:
1. Backache.
2. Foot and Hand Injuries.
3. Recent Advances in Fractures.
Evening. Cocktail Party.

Sunday 14 *August*

9—12 a.m. 1. Common Urological Problems in General Practice.
2. Indications for the Surgery of Mitral Disease.
3. Result of Surgery of Pulmonary Tuberculosis.
2—5 p.m. Modern Trends in Peripheral, Arterial and Venous Disease.

Fee for the course is £4 4s. 0d. which includes the University Registration of £1 0s. 0d.

All lectures will take place in the Lecture Demonstration Theatre (Room 45), Department of Surgery, Medical School, Johannesburg (unless otherwise stated).

This course is expected to be popular, and so early application is advisable.

* * *

The first golf competition for a trophy presented by Abbott Laboratories, to be competed for annually by doctors in the Free State and northern Cape Province, will take place on the course of the Bloemfontein Golf Club on 11 July 1955. All entries should reach Dr. P. Connan, 221-6 United Building, Maitland Street, Bloemfontein, by 29 June, and all communications should be forwarded to Dr. Connan.

CONGRESS NEWSLETTER : KONGRESNUUSBRIEF

To all Members of the Association:

A further circular giving details of the Congress to be held in Pretoria from 17 to 22 October 1955 will be mailed to members towards the end of this month and it will contain additional information regarding the various activities of Congress.

We are pleased to announce that Their Excellencies the Governor General the Honourable Dr. E. G. Jansen and Mrs. Jansen have been so gracious as to extend an invitation to members of Congress to attend, with their wives (or husbands), a reception at Government House on Tuesday afternoon 18 October, at 5 o'clock.

Another feature of Congress week is an organized tour of the Kruger National Park during the week-end 22 to 24 October. During this period the Game Reserve is usually closed, but the National Parks Board have kindly agreed to open it for members of Congress and their wives. It will be a conducted tour at a cost of approximately £2 10s. per head (excluding transport). The number that can be accommodated is limited and those desirous of availing themselves of this opportunity should contact the Organizing Secretaries immediately and at the same time indicate whether they will be requiring transport or have their own cars.

The Pretoria Rotary Club holds its weekly meeting at the Hotel Assembly every Thursday at 1 o'clock. It will take place as usual on Thursday, 20 October during Congress week. Members of Congress who are Rotarians and intend being present at this luncheon are kindly advised to communicate with Rotarian Waks.

Arrangements are being made so that members who wish to do so may be able to attend Race Meetings during Congress week. Those members who wish to participate in the various sporting events associated with Congress are requested to submit to the Organizing Secretaries their names and the particular sport in which they are interested.

We should again like to stress the necessity for making early hotel reservations. The number of hotel bookings already made is far short of the number of intention cards received and it is quite possible that members have forgotten to reserve their accommodation. Please do this immediately to avoid disappointment, as hotel accommodation in Pretoria is already becoming very difficult.

C. M. Grundlingh and W. Waks
Hon. Organizing Secretaries

Aan alle Lede van die Vereniging:

Teen die einde van hierdie maand sal 'n verdere omsendbrief met besonderhede i.v.m. die Kongres wat op 17-22 Oktober 1955 te Pretoria gehou word, aan lede gepos word. Dit sal addisionele inligting omtrent die verskillende bedrywighede van die Kongres bevat.

Ons kondig met genoeë aan dat hul Eksellensies, die Goewerneur-Generaal dr. E. G. Jansen en mev. Jansen 'n vriendelike uitnodiging tot 'n onthaal rig aan lede van die Kongres en hulle gades. Die resepsie vind plaas te Goewermentshuis op Dinsdag, 18 Oktober om 5 nm.

Rëelings is getref om oor die naweek, 22-24 Oktober, 'n georganiseerde toertjie van die Kruger-wildtuin te maak. Die Wildtuin is gewoonlik gedurende hierdie seisoen gesluit, maar die Nasionale Parkeraad het vriendelik ingewillig om die Wildtuin te open vir Kongreslede en hul gades. Dit sal 'n begeleide toer wees, teen 'n koste van £2 10s. per persoon. (Reiskoste word nie by hierdie som ingesluit nie.) Aangesien slegs 'n beperkte tal lede kan meedoen, moet dié wat van die geleentheid gebruik wil maak onmiddellik met die Organiserende Sekretarisse in verbinding tree. Die bestuur moet terselfdertyd in kennis gestel word of lede hul eie motors sal gebruik, of wil hê dat vervoer gereël moet word.

Die Rotariërklub, Pretoria, hou hul vergadering weekliks om een-uur by die Hotel Assembly. Soos gewoonlik vind dit gedurende Kongresweek op Donderdag, 20 Oktober plaas. Rotariërlede van die Kongres moet Rotariër Waks daarvan verwittig as hul die noenmaal wil bywoon.

Rëelings word ook getref vir lede wat die perdewedrenne gedurende Kongresweek wil bywoon. Verskeie sportnommers vind oor die Kongresweek plaas, en belangstellende lede moet die Organiserende Sekretarisse van hul name verwittig asook van die sport waaraan hulle wil deelneem.

Ons wil nogmaals die noodsaaklikheid van vroegtydige hotelreservasies benadruk. Vergeleke met die aantal aannemingskaartjies wat ontvang is, is baie min besprekings gedoen. Moontlik het die kwessie lede ontgaan. Om teleurstelling te voorkom wil ons by u aandring om hierdie saak u onmiddellike aandag te skenk—akkommodasie in Pretoria is nou al reeds moeilik te vinde.

C. M. Grundlingh en W. Waks
Organiserende Ere-Sekretarisse

BOOK REVIEWS : BOEKRESENSIES

YEARBOOK ON TREATMENT

Modern Treatment Yearbook 1955. Edited by Sir Cecil Wakeley, Bt., K.B.E., C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S. Twenty-First edition. Pp. 344 +xxii with 45 illustrations. 25s. London: Baillière, Tindall & Cox, Ltd. 1955.

Contents: Antibiotics in Medicine. 2. Antibiotics in Surgery. 3. Antibiotics in Neurology. 4. Antibiotics in Skin Diseases. 5. Antibiotics in Tuberculosis. 6. Antibiotics in the Treatment of Venereal Disease. 7. The Newer Antibiotics in Ophthalmology. 8. Antibiotics in the Treatment of Urinary Infections. 9. The Emergence of Antibiotic-Resistant Strains of Bacteria. 10 Modern Treatment of the Injured Foot. 11. The Modern Treatment of Hepatitis. 12. The New Insulins. 13. Antiseptics in General Practice. 14. Intractable Pain. 15. Bornholm Disease. 16. Some Soft-Part Lesions of the Forearm and Hand. 17. Modern Treatment of Bronchopneumonia in Infants and Young Children. 18. Modern Treatment of Injuries of the Hand. 19. Hyperemesis Gravidarum. 20. Rheumatic Heart Disease in Children. 21. The Treatment of the Failing Heart. 22. The Investigation and Treatment of Obesity. 23. The Modern Treatment of Skeletal Tuberculosis. 24. The Use of Supervoltage Therapy in the Treatment of Cancer. 25. The Medical Treatment of Hypertension. 26. Obliterative Arterial Disease of the Legs. 27. Blood Vessel Grafting. 28. Diseases of the Supporting Tissues of the Teeth. 29. Myasthenia Gravis. 30. Temporal Arteritis: Its significance and treatment. 31. The Industrial Approach to Rehabilitation. 32. 'Growing Pains'. 33. The Parotid Gland. 34. The Modern Treatment of Depressive States. 35. Modern Treatment of Adrenal Deficiency Disorders. 36. Prolonged Labour. Index.

One year book is very like another. This one is particularly undistinguished. It is also remarkably out of date considering that it is a 1955 publication. For instance: (1) there is almost nothing on the use of isomiazid in the chapter on tuberculosis, while it is not even mentioned in the section on tuberculosis meningitis; (2) hypertension has got as far as hexamethonium bromide, but rauwolfia veriloid, apresoline and ansolysin are not considered; (3) largactil has not appeared, e.g. in hyperemesis gravidarum; (4) glutamic acid is still recommended for hepatic coma, while the blood ammonia is not mentioned, and nor is cortisone in this connection. Liver extract is still given for cirrhosis.

As usual there are far too many vague statements, too many drugs or methods 'may be used' for this or that, without any explanation or discussion. Dr. Schlesinger's chapter on rheumatic heart disease is good, and the one on supervoltage therapy interested your reviewer. Some are bad, as that on the failing heart, which is best unread.

Bornholm disease is a strange choice—5 pages, with 6 lines on treatment!

This book is neither stimulating nor always sound and can only be recommended to chronic yearbook devotees.

W.P.U.J.

SURGERY FOR NURSES

Modern Surgery for Nurses. Edited by F. Wilson Harlow, M.B., B.S. (Durham), F.R.C.S. (Eng.). Third Edition. Pp. 872+xxiv with 419 illustrations. 27s. 6d. London: William Heinemann Medical Books Ltd. 1954.

Contents: 1. Haemorrhage and Surgical Shock. 2. Saline and Dextrose infusion and blood and plasma transfusions. 3. Wounds and superficial injuries. 4. Acute inflammation and infective gangrene: Infections of the hand. 5. Chronic inflammation: Tuberculosis, syphilis and actinomycosis. 6. Surgical Bacteriology. 7. Tumours and Cysts. 8. Ulcers, sinuses and fistulae: Skin-grafting. 9. Burns and scalds. 10. Diseases of the veins and arteries: Gangrene: Amptations. 11. Peritonitis. 12. Appendicitis, and diseases simulating appendicitis. 13. The oesophagus, stomach and duodenum. 14. The Gall-bladder and bile ducts, liver, pancreas and spleen. 15. Hernia and intestinal obstruction. 16. The rectum and anal canal. 17. Urinary cases: Special instruments and diagnostic investigations. 18. The kidneys and ureters. 19. The bladder and prostate. 20. The urethra and male genital organs: The adrenal glands. 21. Thoracic surgery. 22. The Breast. 23. The neck and thyroid gland. 24. The face, salivary glands, mouth, tongue, and Teeth. 25. The throat, nose and ear. 26. Head injuries and neurosurgery. 27. The examination of urine and other diagnostic tests in common use. 28. X-ray diagnosis. 29. Preparation for operation and methods of anaesthesia. 30. Surgical technique. 31. Post-operative treatment. 32. Post-operative complications. 33. The care and treatment of special cases. 34. Fractures, dislocations and other injuries to bones and joints. 35. Injuries of individual bones and joints, upper limb and chest. 36. Injuries of Individual bones and joints, lower limb, pelvis and spine. 37. Injuries and diseases of burase, muscles and tendons. 38. Inflammatory disease of bones and joints. 39. Deformities, congenital and acquired. 40. Affections of the spine. 41. Nerve lesions. 42. Tumours and non-infective diseases of bone. 43. Gynaecological anatomy: Normal and abnormal menstruation. 44. Diseases and displacements of the uterus. 45. Pregnancy and labour and their anomalies and complications. 46. Infections of the genital tract and diseases of the vulva and vagina. 47. Cysts and tumours of the ovaries. Sterility. Gynaecological instruments and pre- and post-operative treatment. 48. Ophthalmic surgery and nursing. 49. Syphilis, gonorrhoea and other venereal diseases. 50. General anaesthesia. 51. Intravenous anaesthesia: Basal narcosis and rectal anaesthesia. 52. Spinal analgesia: Local, regional and epidural analgesia. 53. Radiotherapy in the treatment of malignant tumours: Surgical electrical instruments. 54. Useful tables and clinical details. 55. The sulphonamides and antibiotics.

This is a volume of the utmost comprehensiveness, dealing with all branches of Surgery and in addition Gynaecology, Obstetrics, Anaesthetics, Surgical Bacteriology, Diagnostic and Therapeutic Radiology and antibiotic and sulphonamide Chemotherapy.

The reader is first impressed by the systematic lay-out of the text and welcomes the larger-than-average print. Heavy print is used to great advantage and aids in simplifying classification and tabulation, thereby contributing to easy reading and reference.

The many clear photographs and diagrams have been effectively chosen to give the nurse not only a clear clinico-pathological picture but also to illustrate the main operative procedures and optimal positioning of the patient at all stages of nursing.

The reader will profit by the special chapters outlining the all-important pre-anaesthetic and surgical preparations and the nursing surveillance during the post-operative period with its hazardous complications, plus their recognition and emergency treatment.

A section on the essentials of paediatric nursing and the diabetic patient is more than welcome. One is, however, acutely aware that no mention is made of the essentiality and great dangers of parenteral-fluid therapy in major surgery, particularly of infants.

Both the theatre-trained and the trainee will view with delight the chapter on theatre technique. This is also amply complemented in most chapters by drawings of surgical instruments with their names and uses aptly described in the captions.

Not a single X-Ray photograph included is too technical for the nurse. Being carefully selected they aid her in clarifying the underlying pathology of her patient and thus the rationale of the prescribed treatment which she is taught to carry out.

One is amazed to find a volume of this size and scope where the editor has so successfully maintained brevity of the text in every section without sacrificing clarity or surgical matter essential for the practising nurse.

The above-mentioned are but a few of the many qualities which contribute to make this volume invaluable, not only for study and examination preparation, but also as a quick reference for the ever-busy nurse.

P.W.M.

LABORATORY TECHNIQUES IN RABIES

Laboratory Techniques in Rabies. By various authors. World Health Organisation: Monograph Series, No. 23, 150 pages; 51 illustrations (including 1 coloured plate), price £1 (paperbound, or £1 5s. clothbound). South African Agent: Van Schaik's Book Store (Pty), Booksellers and Stationers, Pretoria.

Contents: Foreword. 1. Laboratory diagnosis. 2. Methods of vaccine production 3. Vaccine potency tests. 4. Production of hyper-immune serum. 5. Laboratory. Additional reference sources.

With the publication of this useful manual, the World Health Organization has fulfilled a need for information on new laboratory techniques of rabies, as well as combined in one volume all the basic procedures necessary for laboratory work.

The manual is on the whole very easy to follow, and each step, is clearly set out, from the preparation of a brain emulsion and the inoculation of mice for testing for the presence of the virus to the preparation of a vaccine and its final testing for sterility and potency. Many excellent photographs are included.

All the contributors are experts in the field of rabies. They have selected reliable procedures with the intention of introducing methods which would aid in producing more uniform and comparable results in laboratories engaged on this work. However, they have not forgotten that the facilities in many laboratories are limited; for instance, in the preparation of vaccine, more than one method is described, the first being the preparation of a phenolized vaccine from sheep brains, and the second one in which rabbit brains are used. Four potency tests, from which any laboratory should be able to select a suitable method, are also given. In addition, the preparation of a chicken-embryo vaccine and its potency test are described. One rather confusing mistake occurs in the method of preparation of a diluent for a brain suspension. Incorrect proportions of the required constituents are given;

apparently the contributor has failed to check his figures. In the section on the production of hyper-immune serum, very little information has been given and the methods used are not too clear.

Many of the techniques for rabies work that are given in this manual, can also be applied to other viruses.

The contributors are Drs. D. d'Antona, P. Atanasiu, R. Béquignon, E. Falchetti, Karl Habel, George A. Hottle, Harold N. Johnson, Martin M. Kaplan, Kamarov, Hilary Koprowski, Pierre Lepine, Thomas F. Sellers, Ernest S. Tierkel and C. Vialat.

N.W.

BOOKS RECEIVED : BOEKE ONTVANG

Fractures and Joint Injuries. Volume II, Fourth Edition. By Sir Reginald Watson-Jones, F.R.C.S., F.R.A.C.S. (Hon.), F.A.C.S. (Hon.), F.R.C.S.E. (Hon.), M.Ch.Orth., B.Sc., M.B., Ch.B., M.R.C.S., L.R.C.P. Pp. 1073 + viii, with 1613 illustrations. £6. Edinburgh and London: E. & S. Livingstone Ltd. 1955.

Roentgenographic Anatomical Terminology. Its Origin and Definition. By J. F. Roderick, R.T. Pp. 20. New Jersey: E. I. du Pont de Nemours and Co. (Inc.).

Bickam-Callander Surgery of the Alimentary Tract, Vols. I, II and III. By Richard T. Shackelford, M.D. Pp. 2575, with 1705 illustrations. £21 12s. per set of 3 vols. Philadelphia and London: W. B. Saunders Company. 1955.

Mitchell's Anatomy and Physiology, and Causes of Disease. Third Edition. By H. C. Trowell, O.B.E., M.D., F.R.C.P. Pp. 244 + xvi, with 87 illustrations. 7s. 6d. London: Baillière, Tindall and Cox Ltd. 1955.

Principles of Medical Statistics. Sixth Edition. By A. Bradford Hill, C.B.E., D.Sc., Ph.D., F.R.S. Pp. 314 + ix. 10s. 6d. London: The Lancet Ltd. 1955.

Communicable Diseases. Third Edition. By Franklin H. Top, A.B., M.D., M.P.H., F.A.C.P., F.A.A.P., F.A.P.H.A. Pp. 1208, with 109 illustrations. £7 17s. 6d. St. Louis: The C.V. Mosby Company. 1955.

The Management of Obstetric Difficulties. Fifth Edition. By Paul Titus and J. Robert Willson, M.D., M.S. Pp. 737, with 348 illustrations. £5 6s. 3d. St. Louis: The C.V. Mosby Company. 1955.

British Postgraduate Medical Federation, University of London. Lectures on the Scientific Basis of Medicine. Volume III, 1953-1954. Pp. 366 + ix, with 7 illustrations. 35s. London: University of London, The Athlone Press. 1955.

The Physiological Basis of Medical Practice. A Text in Applied Physiology. Sixth Edition. By Charles Herbert Best, C.B.E., M.A., M.D., D.Sc. (Lond.), F.R.S., F.R.C.P. (Canada) and Norman Burke Taylor, V.D., M.D., F.R.S. (Canada), F.R.C.S. (Edin.), F.R.C.P. (Canada). Pp. 1357 + xiii, with 601 illustrations. 91s. 6d. London: Baillière, Tindall and Cox Ltd. 1955.

Diseases of the Liver and Biliary System. By Sheila Sherlock, M.D. (Edin.), F.R.C.P. (Lond.). Pp. 720 + xv with 198 illustrations. 50s. Oxford: Blackwell Scientific Publications. 1955.

The Year Book of Dermatology and Syphilology (1954-1955 *Year Book Series*). Edited by Marion B. Sulzberger, M.D. and Rudolf L. Baer, M. D. Pp. 472, with 59 illustrations. 86·00. Chicago: Year Book Publishers, Inc. 1955.

CORRESPONDENCE : BRIEWERUBRIEK

ANAL FISSURE

To the Editor: In the *Journal* of 28 May 1955 there was a letter[1] discussing the treatment of anal fissure.

I should like to stress that my paper in the Revision Series *The Surgery of Ano-rectal Conditions including Haemorrhoids*[2] was written mainly for the general practitioner. At present I am not disputing that internal sphincterectomy is efficacious in anal fissure, nor am I disputing the fact that in time it may become the standard operation for this condition. However, I feel that the operation of internal sphincterectomy is outside the scope of the general practitioner. Moreover, in my personal practice and in the practice of others with whom I am associated, we can justifiably claim good results for the operation of excision of the fissure and external pile and sectioning of the subcutaneous sphincter. The importance of daily passage of the St. Mark's dilator for 2 weeks must not be forgotten.

S. Skapinker

924 Philadelphia Corner
Jeppe Street
Johannesburg
8 June 1955

1. Eisenhammer, S. (1955): S. Afr. Med. J., **29**, 532.
2. Skapinker, S. (1955): *Ibid.*, **29**, 407.

TREATMENT OF PRURITUS ANI

To the Editor: An interesting paper by Mr. Samuel Skapinker[1] on the surgery of the ano-rectal region published in the *Journal* of 30 April has prompted me to give my experience in the treatment of pruritus ani, a very common and distressing complaint. The treatment I have found effective is very simple and almost invariably gives relief. The usual complaint is itching round the anus coming on at night—seldom during the day.

On examining these patients one finds the skin round the anus is moist—worse at the end of the day, as is to be expected after the exertions of the daily routine. The skin is generally thrown into folds and is probably reddened and angry-looking from being scratched.

Early in my experience it struck me that the common cause for this irritation was either a fungus or organism similar in nature to a yeast, and that at the end of the day the organism became active in the moist warm secretion which is a suitable nidus for activity.

Whatever the cause I seldom failed to relieve the patient by the simple method of local cleanliness. In the rare case in which there is an underlying systematic cause, such as diabetes, worms in children, food allergy, etc., this factor must be treated, but the local treatment is equally important. The advice given to the patient was as follows:

1. Before retiring to rest go to the bath-room and with a soft cloth, using plenty of soap, wash the parts thoroughly in a manner resembling almost the ritual of the surgical scrub-up of the hands. In very severe cases one may advise after the washing with soap and water the application of 1 in 40 carbolic lotion; this, however, is seldom necessary. This simple procedure almost invariably gives the patient a night's rest.

2. If the patient is wakened at night with an attack of itching, let him get up and go through the same proceedure, washing carefully with soap and water. Again the relief is almost invariable.

Patients have been referred to me for operation. They have been surprised and at first rather sceptical at the advice I have given them—soap and water—and I was rather ashamed at charging a consultation fee for such simple advice. These patients almost invariably came back and reported great relief and satisfaction. Ointments in my opinion are contra-indicated; they seal up the folds and tissues which lodge the offending cause—organism or fungus?

If operation is carried out (it is seldom necessary) all that is usually required is to strip away the rugi and folds of skin, leaving a smooth surface, *plus* keeping the parts clean with soap and water. This generally brings relief. If piles or a fissure or any other local condition is present operation may be necessary to cure this complicating factor.

In no surgical book — and I have consulted many — has this simple treatment been recommended. After almost 50 years of surgical practice one picks up a few simple therapeutic tips. I hand this one on.

Lennox Gordon

1. Skapinker, S. (1955): S. Afr. Med. J., **29**, 407.